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THE FORAMEN OF RIVINUS—AN ARTEFACT

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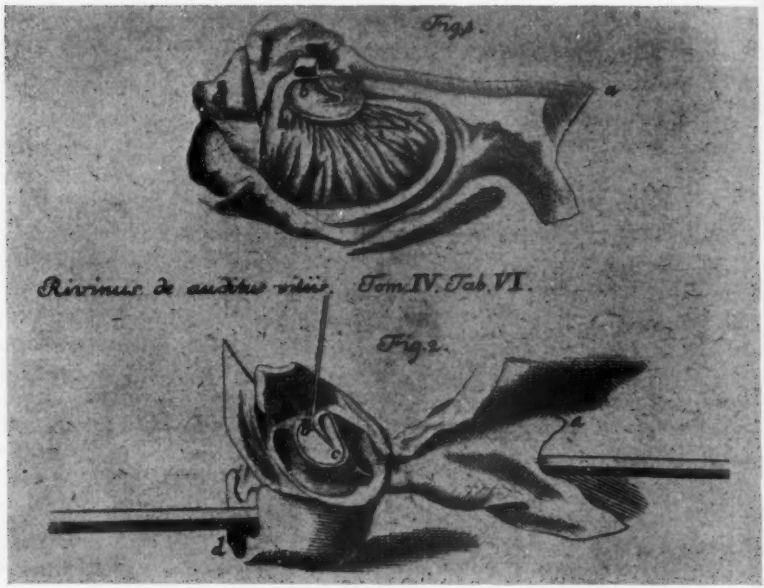
IN 1717, Rivinus submitted his inaugural medical dissertation at Leipzig. He chose the subject "De auditus vitiis"—concerning faults of hearing. Much of the matter of this thesis is taken up with a description of a small hiatus of the tympanic membrane, that he had been able to demonstrate 28 years earlier. It was not an entirely new discovery and the presence of a passage through the tympanic membrane had been inferred from early times. Alcmaeon of Crotona writing around 450 B.C. had declared that goats were able to breathe through their ears. It was common experience that some people were able to make air whistle out of the ear when the nose was blown violently. The curious case of a lady who could extinguish a candle held close to the ear by performing what was to become known as the Valsalva manœuvre had created wonder and speculation. The latest American craze of smoking had led to the trick of blowing smoke out of one's ears. This evidence caused many anatomists to seek a passage from the mouth to the external auditory meatus. The way seemed clear through the pharyngo-tympanic tube, but only as far as the middle ear. In 1684, Schelhammer concluded his "Treatise on the Hearing Organ" by stating that he personally was unable to demonstrate a passage through the tympanic membrane, adding modestly that it would eventually be shown by one whom Nature had endowed with greater ability and experience.

Rivinus took up this challenge: "I studied the organs of hearing by dissection. At length, in the year 1689, in the month of September, I found the opening that I had long sought, it was in the tympanic membrane

itself, which you see to be displayed by a stylus (see figure). When the rocky portion (the petrous bone) is carefully removed so that the tympanic membrane comes into view, or what is safer, if the hollow bone (the middle ear) is cut through, there will appear within the membrane an irregularity, which shows an open gap when a breath is sent through the tube (the pharyngo-tympanic tube) and which disappears by closing up



Figure—from De Auditus Vitiis-Rivinus 1717. The stylus shown in Fig. 2 passes through the foramen. The specimen is probably that of a sheep.

again as soon as the breath ceases. It easily permits the passage of a fine tube or rod into the auditory canal." Rivinus continues: "It was surprising how closely the fibres gripped the rod, whilst it was being withdrawn, it was like a sphincter closing the aperture when the rod was removed . . . There is no further doubt but that this passage is real and natural from the mouth and nostrils into the auditory meatus." The illustration is evidently not that of a human but some animal, perhaps a sheep. At a later point he adds that he has demonstrated the foramen in the head of a calf and in a sheep of middle age, but that the demonstration of a similar passage in the human must be left to another who is allowed to perform such a dissection.

This account was followed by more reports. In November 1696, Munnicks dissected a human head and was able to demonstrate the foramen

to students and advised the use of a pig's bristle to show its valvular nature. Teichmeyer (1727) found the foramen to be situated close to the chorda tympani where the handle of the malleus joins the head. Cheselden appears to place the hiatus in the pars flaccida. Rudiger found multiple foramina. Valsalva was impressed by an opening at the lower end of the handle of the malleus, which he cautiously observed to be unnatural and an artefact: "Wherever Nature has left a foramen it is closed by a sphincter as is the anus."

A foramen in the tympanic membrane, as described by Rivinus, could not be confirmed by all anatomists. Walther, Heister, Cassebohm and Morgani, amongst others, spoke out against it, but a definite conclusion could not be reached. A direct view of the living tympanic membrane was not yet possible on account of the difficulties of illumination and it was necessary to examine specimens removed from the skull, which were often macerated or dried.

Much earlier in 1709, Ravius had tackled the problem by employing an interesting experimental technique. He filled the middle ear cavity with mercury by injecting it down the pharyngo-tympanic tube, and demonstrated that it flowed out through the external auditory meatus. Unfortunately few details of the procedure were given, but it seemed to be an experiment that was worth repeating. A cat's head was employed and an identical result was forthcoming. But further tests showed that the passage of mercury through the tympanic membrane was associated with the height of the column of mercury and the degree of maceration of the specimen rather than the presence of any valvular foramen. Huschke, writing in 1824, denied the existence of a valvular opening or a patent canal as a normal structure. In certain circumstances a foramen might be caused by a pathological process or possibly an abnormality or failure of development of the embryo which he termed "inhibition formation"—*Hemmungsbildung*. In very young embryos he believed that the auditory canal was open in the superior part and that the tympanic membrane might be entirely lacking in the first periods of embryonic life when the auditory canal and the eustachian tube might form an open continuous passage which he considered to be homologous with the first branchial cleft.

Stimulated by a report by Fleischmann (1836) of a Rivinian foramen in the mole, and the bats: Vespertilio and Rhinolophus, Hyrtl (1845) reinvestigated the whole problem and examined his collection of seventy human and mammalian ears with meticulous thoroughness. He was unable to find the least evidence of a valvular foramen as described, and observed that the tympanic membrane, if preserved for a long time, tended to become friable, and that a tear might develop in a characteristic position in the membrane posterior to the handle of the malleus. He accounted for the Rivinian foramen by this artefact.

This convincing evidence by one of the most careful investigators has not been accepted by all anatomists. The publication of accounts of the foramen of Rivinus continues. It is described in different positions (Bochdalek, 1866) embellished by valvular flaps and running an oblique course through the substance of the membrane. Several latest editions of standard textbooks on otology (Ballenger, Eggston and Wolff, Scott-Brown) continue to give space to its description without any convincing evidence. A recent monograph on the middle ear (Kobrak, 1959) sums up a discussion on the existence of this foramen as "not proven".

The presence of a valvular foramen passing from the tympanic cavity to the external meatus, even if present only as a rarity in normal human subjects would seem of such interest that it deserves further investigation. A small foramen in the postero-superior part of the tympanic membrane which might allow the ingrowth of squamous epithelium is suspect in the search for the origin of cholesteatoma of the middle-ear cleft. Habermann's statement that keratinizing squamous epithelium can grow through such a perforation of the tympanic membrane in the same fashion as a fistulous track or sinus can become lined with squamous epithelium is now generally accepted and the process has been demonstrated in the experimental animal. This process is now widely acknowledged as the probable explanation of most cholesteatomas.

Many new techniques of investigation have become available since Hyrtl's pronouncement and it would now seem possible to reach a definite conclusion concerning the existence of the Rivinian foramen.

Evidence from Clinical Experience

The overcoming of the problems of illumination and the introduction of the aural microscope enables the very careful examination of the tympanic membrane under magnification to be carried out as a routine. But no reports of the foramen in the recent literature are known.

Evidence from Anatomical Studies

The tympanic membrane can be subjected to the most careful scrutiny by the examination of serial microscopical sections. Routine examination of every tenth section cut at 20 mm. thickness of the temporal bone of human and experimental animal has not yielded reports of any structure suggesting a Rivinian foramen.

Evidence from Developmental Anatomy of the Mammalian Tympanic Membrane

At the 9th week of embryonic development (36 mm.), the meatal plate is a solid ingrowth of ectodermal cells extending from the first external groove, lying inferiorally and laterally, towards the endoderm lining the

outer part of the tubo-tympanic recess. These layers are separated by a thick layer of mesenchymal tissues which contains the handle of the malleus in its cartilagenous form. The tympanic ring (1·5 mm. in diameter) is faintly indicated as a concentration of mesenchymal tissue. There is no other feature of the tympanic membrane region at this stage.

At the tenth week (50 mm.) the core of ectodermal cells of the meatal plate is beginning to break down and simultaneously the expanding tympanic cavity becomes more closely related to the ectodermal plate. By the 16th week (126 mm.) the development of the tympanic membrane and its supporting ring is well advanced and rapidly increasing in diameter and by the 23rd week (210 mm.) it is approaching its mature size, but the intermediate mesenchymal layer remains as featureless fibrous tissue.

The fusion of the tympanic ring to the squama, the lodgement and fixation of the fibrous layer in the sulcus tympanicus, the differentiation of the pars flaccida and tensa and the gradual change in the plane of the tympanic membrane caused by the differential growth of the tympanic ring bring about the mature form of the tympanic membrane. These modifications in form and changes in relationship are more complicated than described in the standard accounts (Anson *et al.*, 1955). Study of these later stages has not furnished evidence of any normal or vestigial structures within the tympanic membrane that might account for the Rivinian foramen.

Anomalies of this region are rare. They are invariably atresias, usually extensive. The region of the tympanic membrane remains a thick and solid barrier between the external auditory meatus and the middle-ear cleft. Congenital absence of the tympanic membrane is unrecorded and there is no evidence to support the embryological hypothesis of Huschke.

Evidence from the Comparative Anatomy of the Mammalian Tympanic Membrane

Opinion on the evolution of the mammalian middle ear and the tympanic membrane has been divided. Gaupp (1912) believed in a different method of evolution of the reptilian and mammalian ear cavity, the former developing above, the latter below the mandible in such a way that the one could not have arisen from the other. Goodrich (1930), however, held that the reptilian and the mammalian eustachian tube, middle-ear cavity and tympanic membrane were completely homologous with one another. Shute (1956) has recently put forward an alternative theory which takes into account various objections to these older explanations. He has suggested that the mammalian middle ear has evolved either from the submandibular sulcus and lateral diverticulum of the floor of the pharynx described in lizards, or more probably by the incorporation of the original tympanic cavity into the submandibular sulcus. If this latter alternative is accepted, the pars tensa is regarded as an extension of the original

reptilian drum and the pars flaccida a new formation consequent upon the retreat of the body of the malleus and incus into the middle ear.

The study of these evolutionary steps has not furnished evidence or possible explanation of the Rivinian foramen. The claim of Fleischmann that a Rivinian foramen is normally present in moles and bats could not be confirmed by the examination of serial sections of the ears of these animals. No serial sections of a goat's temporal bone has been available for study.

Summary

A valvular foramen of the tympanic membrane, described by Rivinus in 1717 has been the subject of this review, which was suggested by the conflicting accounts of this structure appearing in past and present-day literature. The possible role of such a structure in the formation of middle-ear cholesteatomata seemed worth investigating. The earlier accounts and contemporary discussion has been recalled and from this the true existence of a Rivinian foramen appears extremely remote. Further evidence from the microscopical examination of serially sectioned tympanic membranes, the study of the embryological development of the ear and the evolution of the mammalian eardrum has been undertaken, but no evidence to support the existence of a structure described by Rivinus has been forthcoming. The reports of the foramen in the early literature are apparently concerned with an artefact brought about by maceration and drying of a specimen before its examination. The accounts in present-day text books offer no new evidence to support the existence of the foramen and appear to be a perpetuation of a fallacious observation.

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ACOUSTIC IMPEDANCE AS AN AID TO DIAGNOSIS IN OTOLOGY

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THE acoustic impedance of an object or of any sound-conducting system may be defined as the resistance exhibited by it to a wave of sound energy, and it depends on the vibratory characteristics of the object or the system. In order to measure the acoustic impedance of the ear it is sufficient to measure the amount of absorption of the sound energy by the ear, and the impedance and the absorption may be taken as being inversely proportional to each other, that is to say, the greater the impedance the less the absorption, and *vice versa*. Though for complete and accurate determination of the impedance it would be necessary to take into consideration the phase of the sound waves as well as the amount of absorption, in practice this is not of importance for our purpose. Thus, the acoustic impedance of the ear may be described as the resistance exhibited by the sound-conducting apparatus of the ear to a sound entering the external auditory meatus.

The method of measuring the impedance of the ear, its application to clinical otology and its development as a research tool have been evolved and investigated in recent years by various Danish workers, but not a great deal has been written on the subject in the non-Scandinavian literature. Following the introduction of the principle to clinical work by Metz in 1946, several other workers such as Jepsen (1951, 1953), Kristensen *et al.* (1952), Thomsen (1955) and Terkildsen *et al.* (1959) have developed its various applications and improved the methods and techniques used. It is proposed, therefore, to outline briefly the results of their work in this field.

Measurement of the Acoustic Impedance of the Ear

The measurement of the acoustic impedance of the ear was first applied clinically by Metz, using a mechanical acoustic measuring bridge, and although this has now been superseded in the Danish clinics concerned by electro-acoustic methods the principles involved are most simply described and understood by reference to the original mechanical device.

By means of this bridge the unknown acoustic impedance of the ear is measured by comparing it with a known variable impedance of the bridge. The apparatus (Fig. 1) basically consists of a long straight tube, with one end in the ear, while at the other end there is a device for altering the

impedance in the tube, the alterations being read from calibrations. Exactly in the middle of the tube is a telephone diaphragm which sends sound waves along it in both directions simultaneously, but of course in opposite phase. That is to say, the vibrating diaphragm causes a phase of rarefaction, or trough, on one side as it produces a condensation phase, or peak, on the other. From the main tube two identical short tubes lead off

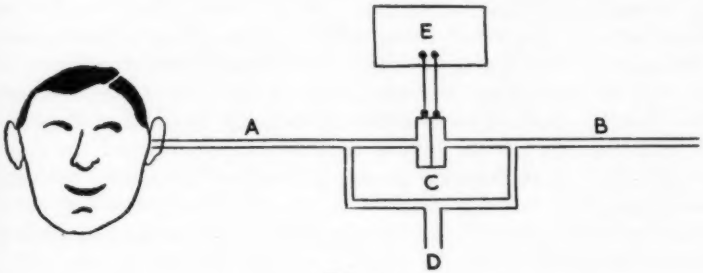

FIG. 1.
The Acoustic Bridge. (After Metz.)
A. End of Bridge in tested ear.
B. Calibrated end of Bridge.
C. Telephone diaphragm.
D. Auscultation point.
E. Oscillator.

symmetrically at equal distances from the telephone diaphragm, and they in turn unite in a single tube known as the auscultation point, to which a stethoscope is attached.

When the variable impedance in the calibrated end of the tube becomes equal to the unknown impedance in the end introduced into the ear, the two sound waves from each end of the tube will reach the stethoscope with the same amplitude but with a phase difference of 180°. Therefore, they will counterbalance each other and the observer will hear no sound at the auscultation point.

If the balance is altered by a change of the impedance either in the ear or in the calibrated bridge, then the examiner again hears the tone through the stethoscope. The size of any alteration in impedance in the ear can be calculated by the change needed in the variable impedance to re-establish the balance, so that the tone disappears again at the auscultation point and is no longer heard by the examiner. We see then that by this method we can measure the impedance of the ear against a known impedance in the bridge, and we can also detect and measure any change in impedance which occurs in the ear. Actually this mechanical bridge, as used by Metz, has not been commercially available. Although the mechanical acoustic principle is simple it has to be made with considerable precision. Therefore, an electro-acoustic bridge has been evolved by Terkildsen

and Scott-Nielsen (1960), working on the same lines as the mechanical one according to the zero balancing principle. In this two identical micro-telephones are connected to the external ear canal by two narrow tubes. The micro-telephones and tubes are built together in a simple ear insert with an inflatable cuff round the inner end to obtain an airtight fit in the meatus. Thus, the deep part of the canal is a hermetically closed space, and its impedance is found as follows: One 'phone producing a test tone of a certain frequency establishes a certain sound level in the canal. Using a constant intensity, the sound level depends upon the impedance of the space, which is influenced by variations in the impedance of the drum and conducting mechanism of the ear. The other 'phone is used as a sound probe which, to obtain high sensitivity, is counterbalanced to a zero state by current from the generator which produced the previous tone passing through units for phase and amplitude control. The degree of counterbalance required is read on a vacuum tube voltmeter, preceded by a selective amplifier, which is gradually introduced as the balancing is being completed.

To calibrate the bridge for absolute measurements, impedances are measured in ideal hard closed cavities of various sizes. Directly measured volumes in cubic millimetres are then used to indicate the impedances. A high impedance is equivalent to a small volume, and *vice versa*. The technical details of the electro-acoustic bridge are, of course, probably beyond the understanding or interest of the majority of clinicians.

Clinical Applications

We come next to the clinical applications of the method of impedance measurement. They can be described under two main headings:

(1) Simple measurement of the impedance of the ear.

(2) Measurement of changes which can be produced in the impedance of the ear.

This heading can be subdivided into two:

(*a*) Changes in impedance produced by stimulation of the intra-aural muscle reflex;

(*b*) Changes in the impedance from alteration of the middle-ear air pressure.

(1) *Simple measurement of the impedance of the ear:*

This is of assistance in the diagnosis of the sequelae of suppurative or chronic adhesive otitis, otosclerosis, severe occlusion of the eustachian tube and patulous tube.

As a general rule loss of hearing due to conductive deafness gives a high degree of impedance, that is the amount of absorption of sound

energy is low. Metz has found that if the absorption in a deaf ear exceeds about 30 per cent. the patient will be suffering from a nerve deafness, as opposed to an obstructive type, in 95 per cent. of cases. In a mixed type of deafness, of course, the amount of absorption will depend upon the proportion of conductive deafness involved, as an affection of the perceptive mechanism does not affect the impedance. Thus, a simple determination of the degree of absorption may give us information about the type of deafness. The diseases which are particularly apt to bring about a slight degree of absorption, *i.e.*, a high impedance, are the sequelae of suppurative or chronic adhesive otitis or severe tubal occlusion and otosclerosis. A patulous eustachian tube on the other hand often gives rise to a marked degree of absorption, or very low impedance.

(2) (*a*) *Changes in impedance produced by stimulation of the intra-aural muscle reflexes:*

In daily routine work, however, the information gained by determination of changes produced in the impedance is usually more valuable than that obtained by simple determination of the degree of absorption.

In a normal person if a loud tone is applied to one ear it produces a reflex muscular contraction of the stapedius and tensor tympani in both middle ears. We can compare this with the pupillary light reflex in the eyes. Previously this intra-aural reflex was only shown occasionally through observation of the stapes tendon through a perforation in the drum or through a very transparent drum. This reflex has been shown to produce a change in impedance in the ear. There is a very short latent period, in the region of a tenth of a second, before contraction occurs, and then the contraction subsides more gradually after the end of the stimulus. An increase in the intensity of the stimulus increases the size of change in the impedance and diminishes the latent period, but this is not of importance in ordinary clinical work.

The method of producing the reflex can be described as follows: A telephone receiver connected with the audiometer is placed over the stimulated ear, and the measuring bridge is inserted into the measuring ear. The variable impedance in the bridge is adjusted so that the tone is not heard at the auscultation point. A tone is then introduced into the stimulated ear, and if the reflex occurs the tone will be heard again at the auscultation point after the short latent period. If the intensity of the stimulus is increased step by step the threshold for the reflex at different frequencies can be shown, and it is found that a stimulus of 70 to 90 decibels is needed to produce a reflex in a normal person.

Demonstration of the phenomenon of Recruitment

This is the first use of combining the method described above for elicitation of the aural reflexes, with measurement of the change in im-

pedance of the ear. If, in a patient with loss of hearing in the stimulated ear, the reflex can be produced by a stimulus lower than 70 to 90 decibels above the threshold of the frequencies concerned, as measured audiometrically, recruitment must be present in the stimulated ear. For example, in a case of deafness amounting to 50 decibels at 1000 d.v. per second, if the reflex is elicited by a stimulus of 90 decibels, i.e., 40 decibels above threshold instead of the normal 70 to 90 decibels above threshold, then recruitment is present. The advantages of this method over Fowler's method are two: Firstly, the method is objective, except that some element of subjectivity arises in determining the threshold of hearing originally by audiometry. Secondly, the method can be used also in bilateral loss of hearing. One disadvantage is that other conditions, apart from lack of recruitment, may inhibit the appearance of the reflex, for example, changes in the drum or the middle ear in the measuring ear. Therefore a positive result is conclusive, but a negative result is not quite so.

Psychogenic Deafness and Malingering

This is the second category in which the combined use of these procedures is of value; it has been dealt with by Jepsen (1953), and Thomsen (1955). If no hearing can be demonstrated by audiometry or by voice tests, the hearing may usually be regarded as being absent from that ear, but if the intra-aural reflex can be elicited in that ear it is proof that the ear is capable of hearing. The reflex arc only goes as far as the pons and is not cortical. It is, therefore, not affected by voluntary or even subconscious cerebral factors. The detection of this reflex by alteration of the impedance of the ear is, we must remember, entirely objective as far as the patient is concerned.

Facial Paralysis

In cases of facial paralysis the presence or absence of the intra-aural reflex on the affected side, as shown by change of impedance, is of value in localizing the facial nerve lesion with reference to site of departure from it of the nerve to the stapedius. The stapedial nerve leaves the facial nerve shortly before the chorda tympani, and the tensor tympani is said to get its nerve supply from the fifth nerve. Some doubt about the action of these muscles exists, but it can definitely be shown that on severing the facial nerve central to the departure of the stapedial nerve the intra-aural reflex cannot be elicited as shown by impedance measurement. Therefore, if the tensor tympani gets its nerve supply from the fifth nerve it must be that this muscle cannot produce a change in impedance. Conversely, it seems that motor fifth nerve paralysis cannot prevent the appearance of the reflex. These facts would seem to throw some doubt on the innervation of the tensor tympani.

In cases of total facial nerve palsy, where the reflex is produced in the

ear of the same side, the lesion can be positively located as distal to the site of departure of the stapedial nerve, but if it is not elicited the lesion is most likely to be central to this point. If it is not elicited, however, it must be remembered that conditions other than facial palsy may abolish the reflex, for example, middle-ear disease in the measuring ear on the opposite side.

Middle-Ear Conditions causing Conductive Deafness

As mentioned above, if sound conduction is diminished absorption is also diminished, therefore, impedance is increased. Usually the same condition which causes conductive deafness abolishes the intra-aural muscle reflex, probably owing to the rigidity of the ossicular chain or the tympanic membrane. This is of particular interest in relation to the early diagnosis of otosclerosis, owing to the negative otoscopic findings in this disease. In otosclerosis the reflex cannot be shown, probably due to fixation of the ossicles, and in addition the impedance is often high, and these features are of considerable help in the diagnosis, especially in early stages of the disease.

(2) (*b*) *Changes in impedance from alteration of the middle-ear air pressure:*

Any change of pressure in the middle ear leading to deviation from the external pressure alters the amount of sound absorption and produces an increase in impedance, unless there are already gross changes in the middle ear or tympanic membrane resulting in great impedance which obscures the change.

The measurement of impedance may therefore be used in assessing the function of the eustachian tube in all cases where otherwise only examination of the tympanic membrane could be used to assess the patency of the tube. The advantage is that it is possible to estimate the middle-ear changes quantitatively. For example, when over-pressure is produced in the middle ear examination of the impedance following swallowing makes it possible to assess the ability of the tube to return the pressure to normal. Also, if obstruction of the tube causes rarefaction of the air in the middle ear, inflation of it so as to rectify the difference in pressure on each side of the drum will diminish the impedance. Likewise, if the tube is inflated so that the middle-ear pressure is greater than the external pressure the impedance will again be increased, and when the patient then swallows, if the tube is functioning normally and able to rectify the difference in pressure, the impedance will again be diminished. With regard to the condition known as patulous tube, it has been shown that if there is very pronounced absorption or low impedance, when the tube is open, it is often not possible to obtain a balanced impedance with the bridge, as the impedance of the ear may change synchronously with the respirations.

By combining impedance measurement with Zöllner's (1942) technique for determining the resistance of the eustachian tube, an accurate analysis of tubal function can be obtained (Thomsen), and it is possible to estimate what pressure in the rhinopharynx is necessary to open the tube during swallowing.

Summary

The acoustic impedance of the ear is defined, and the methods of measuring it by the acoustic bridge developed by Metz and by the later developed electro-acoustic bridge are described. The various clinical applications of the procedure are described under the headings of:

(1) Simple measurement of the impedance of the ear.

(2) Measurement of changes which can be produced in the impedance of the ear:

(*a*) by stimulation of the intra-aural muscle reflex,

(*b*) by alteration of the middle-ear air pressure.

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JUVENILE OTOSCLEROSIS*

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Introduction

THE discovery of conductive deafness in a child leaves the essential problem unsolved and raises additional questions. For convenience, these cases may be divided into 2 principal groups:

Those with a slight hearing loss of approximately 20–40 db., and those with a greater loss of hearing, approximately 40–60 db. of air conduction.

To the first group belong cases of catarrhal and allergic affections of the eustachian tube, of the nasopharynx or middle-ear cavity and beside these also cases of enlargement of adenoids and lymphatic tissue at the orifice of the tube and in Rosenmueller's fossa.

To the second main group with a greater loss of hearing belong congenital fixation or absence of the ossicular chain, cases which have undergone during the postnatal age injuries or operations of the ear and where the drum has stayed otherwise undamaged. And finally belong to this group cases of fixation of the stapedo-vestibular junction on an otosclerotic basis.

In the following I wish to report cases as examples of the above-mentioned groups, together with their audiograms:

CASE I.

Boy 7 years old. Mother reports that the boy has been inattentive for the past 3 years and the teacher complains that he does not follow his lessons properly and frequently fails to respond, when called. For years he has been suffering from a dripping nose and frequently complains of earache, but only once—two years ago—had purulent inflammation of the middle ear. Three years ago adenoidectomy. Otoscopy on both sides without significant pathological changes. Ear drum slightly retracted, no irritation. Lining of the cavity of the nose shows a typical allergic reaction with excess mucus. Audiogram: hearing loss through air approximately

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†From the E.N.T. Department of the Italian Hospital in Haifa, and the E.N.T. Service of the Sick Fund (General Jewish Workers Association) Haifa.

30 db. Conclusion: allergic rhino-pharyngo-eustachitis with an allergic middle-ear reaction. Big improvement of the hearing loss after anti-allergic treatment lasting six months.

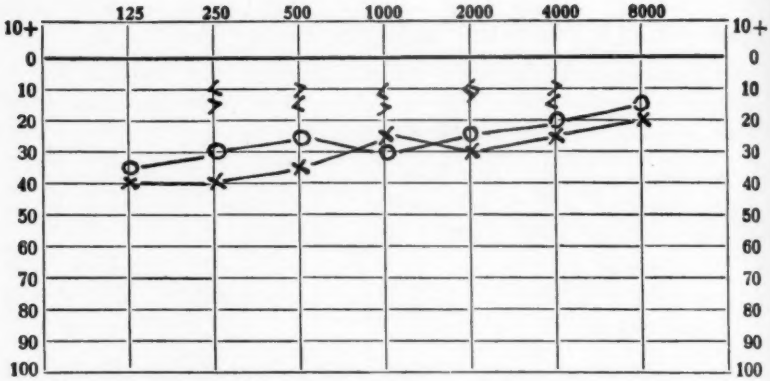

FIG. 1.

Case No. 1. Audiogram of a boy with an allergic rhino-pharyngo- eustachitis and an allergic middle-ear reaction.

air: R: O——O——O bone: R: > >
L: X——X——X L: < <

CASE II.

Boy 5½ years old. Parents report: mouth breathing, poor hearing, often colds. Otoscopically nothing of interest. Nasopharynx full of adenoid tissue. Audiogram: conductive deafness approximately 40 dcb. on both sides. Adenoidectomy performed. Now, one year after operation, hearing almost normal.

CASE III.

Boy 12 years old. Mother reports: bad hearing on left side for past few years. In kindergarten injured by another child in his left ear with the stick of a lollipop. Otoscopy on both sides without pathological signs. Audiogram: conductive deafness left approximately 60 db. Exploration of middle-ear cleft: strong adhesions between promontory and the processus longus incudis, probing stapes and incus fixed. Attempt to free the fibrotic adhesions successful. Middle ear filled up with little pieces of Oxygel soaked in solution of Hydrocortisone. Dexasone per os used one week. Audiogram shows improvement a fortnight after operation. Nine months after operation hearing gap nearly vanished.

Conclusion: traumatic fixation of the ossicular chain. The ear drum shows no pathological changes.

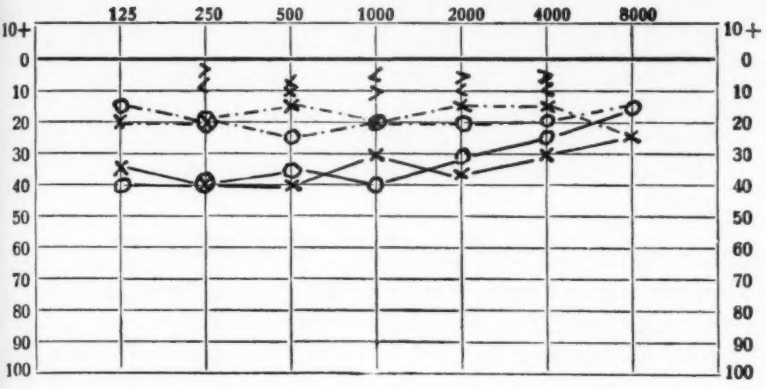

FIG. 2.

Case No. 2. Audiogram of a boy with adenoids in nasopharynx.

air: R: O——O——O preoperat. bone: R: > >
O – . – . – O postoperat. L: < <
L: X——X——X preoperat.
X – . – . – X postoperat.

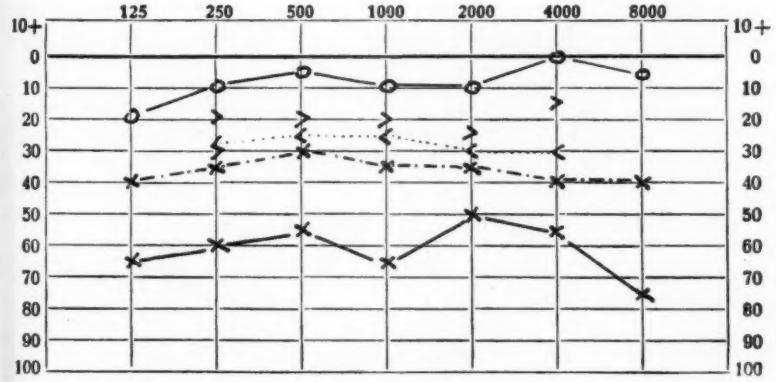

FIG. 3.

Case No. 3. Audiogram of a boy. State after injury of his middle ear: adhesions to the processus longus incudis.

air: R: O——O——O bone: R: > >
L: X——X——X preoperat. L: < <
X—·—·—X postoperat.

CASE IV.

Boy 11 years old. Since childhood bad hearing on both sides, left worse. Repeated audiograms: conductive deafness on both sides. The history reveals no inflammation of the middle ear, there is no pathology of

ear drums and nasopharynx. One year previously adenoidectomy, but no improvement of hearing afterwards. May 1959, exploration of left middle-ear cleft: fibrotic adhesions to the stapes and in the fossa ovalis. Fixation of the stapes. Processus longus incudis absent and replaced by a thin fibrotic cord. Adhesions freed, stapes mobilized and tympanostapediopexy carried out. Improvement of hearing 20–30 db. after operation. Congenital absence of incus with stapes fixation. Case published elsewhere.

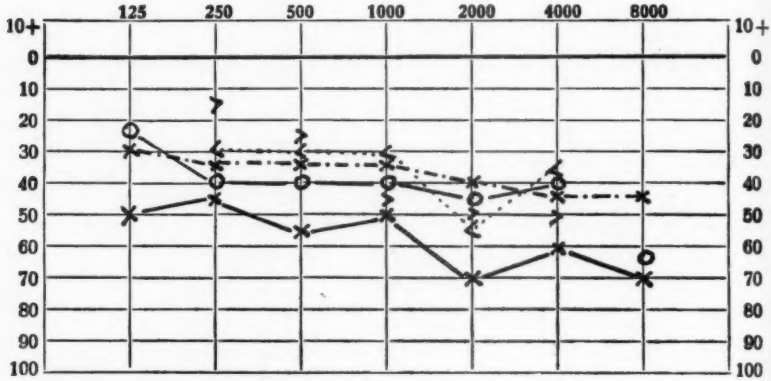

FIG. 4.

Case No. 4. Audiogram of a boy with congenital absence of the incus.

air: R: O——O——O bone: R: > >
L: X————X preoperat. L: < <
X—·—·—X postoperat.

I should now like to bring forward five cases of juvenile conductive deafness, which according to anamnesis, clinical examination, audiograms and exploration of the middle-ear clefts, we are entitled to regard as otosclerotic processes.

Case Reports

CASE I.

W. T., boy 14 years old. Mother reports: bad hearing on both sides for many years, difficulties at school. Family anamnesis for hard of hearing negative. Our diagnosis: chronic inflammatory process left ear, with a perforated drum. Mixed deafness right ear with intact drum. Two years ago tympanoplasty left—Wullstein I. Performed with resulting big improvement of hearing left. December 1959, exploration of the middle-ear cleft right: stapes fixed. Attempt to mobilize fixed stapes without success. Remobilization in June 1960: deep fossa ovalis, in front of the margin of the plate an otosclerotic process. The attempt at indirect

mobilization of the footplate now proved successful and the hearing at once showed marked improvement, which persists until today.

Conclusion: otosclerotic process with stapes fixation right lasting few years. Beginning of hearing loss not precisely known, it probably started during first years of school, approximately between his 6th and 8th year. Audiogram: postoperatively improvement, but persisting weakness of the right VIIIth nerve.

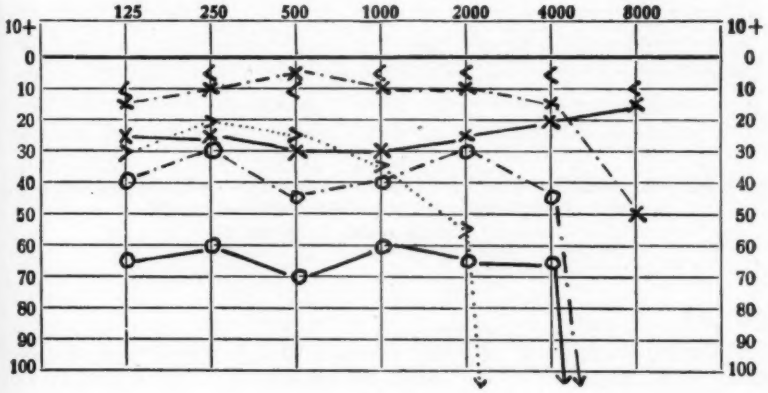

FIG. 5.

Case No. 1 of juvenile otosclerosis. Audiogram of a boy aged 14: otosclerotic stapes fixation right, lasting about 6–8 years. Stapes mobilization right.

Chronic inflammatory process left. Tympanoplasty Wullstein I. left.

air: R: O——O——O preoperat.
O—·—·—O postoperat.
L: X——X——X preoperat.
X—·—·X—·—·X postoperat.

bone: R: > >
L: < <

CASE II.

T. A., girl 16 years old. Bad hearing right since elementary school. No history of middle-ear infections. Nasopharynx, eustachian tube as well as drums without any pathology. Family history negative. Audiogram: conductive deafness right approximately with a hearing loss of 45 db. April 1959, exploration of the middle-ear cleft right: fixation of the stapes, deep fossa ovalis. Indirect mobilization of stapes successful, hearing immediately improved. Audiogram after mobilization: gap vanished. Postoperative repeated controls show till to this day lasting improvemeut of hearing.

Conclusion: otosclerotic process with stapes fixation right, anamnestically lasting from 7 to 9 years of age. Audiogram: conductive deafness. Postoperative improvement of hearing lasting already 1½ years.

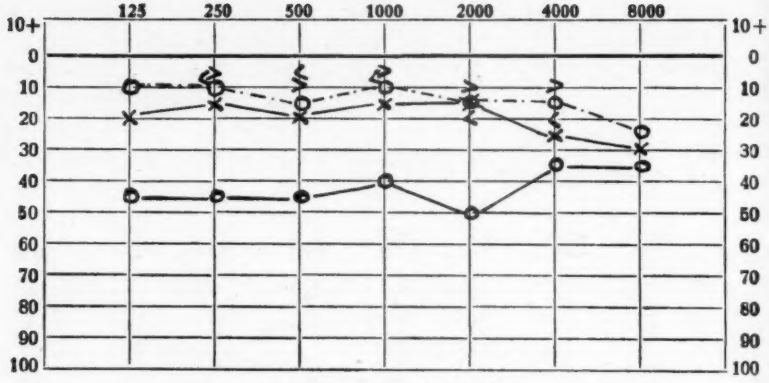

FIG. 6.

Case No. 2 of juvenile otosclerosis. Audiogram of a girl aged 16: otosclerotics tapes fixation right, lasting about 7–9 years. Stapes mobilization right.

air: R: O———O preoperat. bone: R: > >
O—·—·—O postoperat. L: < <
L: X———X

CASE III.

P.L., boy 12 years old, hearing in right ear deficient for the past five years. No family history of otosclerosis. Otoscopy, nasopharynx as well as eustachian tube without pathology. Audiogram: very far advanced mixed deafness right. Hears through air only the deep frequencies of 60–90 db. July 1960, exploration of middle-ear right- stapes fixed, attempt to mobilize indirectly successful, with resulting immediate striking improvement of hearing in the right ear. Sensation of vertigo by probing the mobilized stapes. One month after operation audiogram shows improvement in all frequencies, even in the highest, which he could not hear before mobilization. But at the last examination a fortnight ago, a general deterioration in all frequencies was found, which probably corresponds to an otosclerotic refixation of the footplate.

Conclusion: otosclerotic process with stapes fixation, which probably started round his 7th year. Audiogram: mixed deafness of advanced grade with a remarkable affection of the VIIIth nerve. Mobilization brought big improvement even in the highest frequencies, but this improvement failed to persist after 6 months.

CASE IV.

P.S., boy 13½ years old. Report: bad hearing right for the last 6 years. Never inflammations of the middle ear. Nasopharynx and drums without pathology, family anamnesis for otosclerosis negative. Audiogram: very advanced mixed deafness right. July 1959, exploration of the

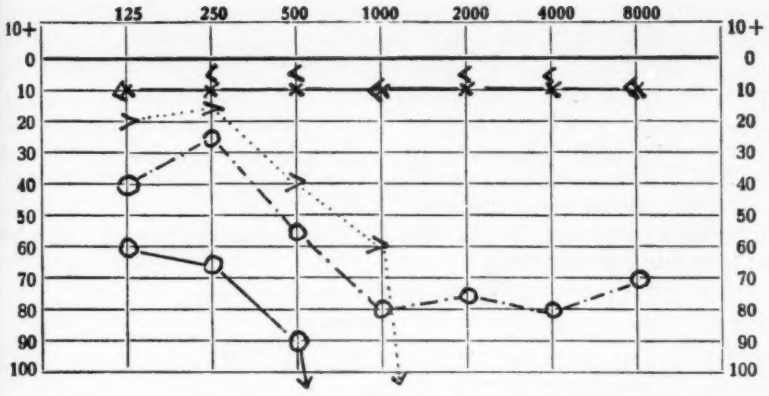

FIG. 7.

Case No. 3 of juvenile otosclerosis. Audiogram of a boy aged 12: otosclerotic stapes fixation right, lasting 5 years. Mobilization right.

air: R: O———O preoperat. bone: R: > >
O—·—·—O postoperat. L: < <
L: X———X

middle-ear cleft right: stapes fixed, the deep and narrow fossa ovalis not clearly seen. Indirect mobilization successful. Audiogrammatically after operation hearing remarkably improved not only through air, but also through bone in the middle and low frequencies. The highest frequencies

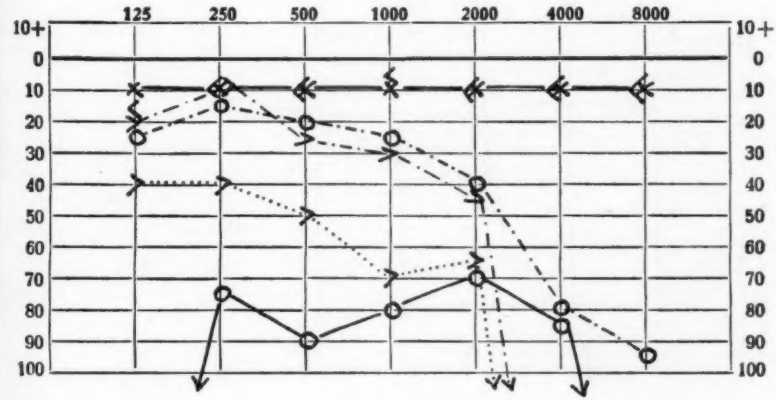

FIG. 8.

Case No. 4 of juvenile otosclerosis. Audiogram of a boy aged 13½ years: otosclerotic stapes fixation right, lasting about 6–8 years. Stapes mobilization right.

air: R: O———O preoperat. bone: R: >·····> preoperat.
O—·—·—O postoperat. >—·—·> postoperat.
L: X———X L: < <

showed no improvement. But approximately 6 months after the operation, hearing starts to deteriorate and at the last examination a fortnight ago, we found the hearing again in the same condition as before the operation, by air conduction as well as by bone conduction.

Conclusion: otosclerotic stapes fixation, anamnestically starting about the 6th or 7th year. Audiogram shows a mixed deafness type of hearing loss with a remarkable nerve participation. It is very interesting to note a big improvement postoperatively not only in the air conduction, but also in the bone curve. Six months after mobilization clinically a refixation of the footplate was observed.

CASE V.

M.J., boy 15 years old. Report: bad hearing left since his 10th year, deteriorating from year to year. Family anamnesis negative. Anamnestically no inflammation of the middle ears. Nose, nasopharynx and ear drums without pathology. Audiograms: very advanced mixed deafness left. August 1960, exploration of the middle-ear cleft: stapes fixation, on the front margin of the footplate an otosclerotic focus. Indirect mobilization successful, hearing improved still on the table. Postoperative audiogram shows improvement of 50–60 db. in the middle and low frequencies through air. Also bone conduction postoperatively improved. Highest frequencies postoperatively still depressed. The hearing improvement persists till today.

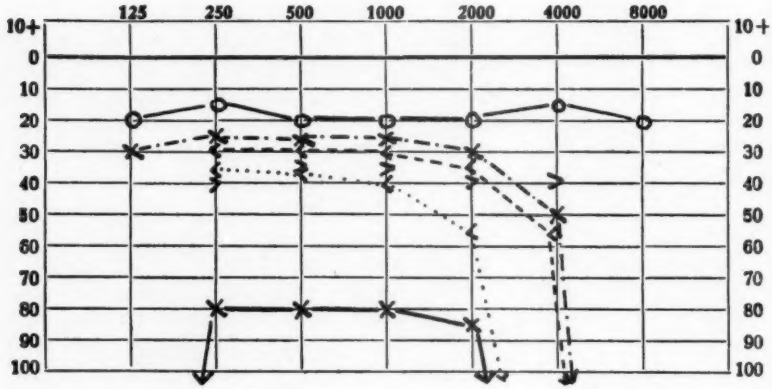

FIG. 9.

Case No. 5 of juvenile otosclerosis. Audiogram of a boy 15 years old.: otosclerotic stapes fixation left, lasting about 5 years. Mobilization left.

air: R: O———O
L: X———X preoperat.
X—.—.—X postoperat.

bone: R: > > >
L: <·····< preoperat.
<—.—.—< postoperat.

Conclusion: otosclerotic fixation of the footplate, which anamnestically started round his 10th year. Audiogrammatically a clear advanced mixed deafness. Postoperative improvement through the air, as well as through the bone!

Summary in our Five own Cases of Juvenile Otosclerosis

(*See the respective table*)

FIG. 10.

Table of survey of our 5 cases of juvenile otosclerosis.

FIVE OWN CASES OF JUVENILE OTOSCLEROSIS

	Sex	First Appearance (age)		AUDIOGRAMATICALLY			POSTOPERAT. COURSE
		at the doctor	of clinic signs	PREOPERAT.	POSTOPERAT. IMPROVEMENT		
					AIR	BONE	
1.	♂	14	6–8	mixed deafness	through all frequencies	no	improvement lasts ¾ year
2.	♂	12	7	,,	,,	no	Refixation clinically and audiometrically after ½ year.
3.	♀	16	7–9	pure conductive deafness	,,	no	improvement lasts 2 years.
4.	♂	13½	6–7	mixed deafness	,,	yes	Refixation clinically and audiometrically after ½ year.
5.	♂	15	10	,,	,,	yes	improvement lasts ½ year.
Total	4 ♂	12–16	6–10	4 mixed deafness	in all cases	in 2 cases	3 lasting improvement
	1 ♀			1 pure conductive deafness			2 refixations

In looking at the above-mentioned five cases, we notice, that there are four boys and one girl.

The patients came the first time to a specialist for examination during their 12th to 16th year. But the history reveals that the clinical otosclerosis with its respective hearing loss started already between their 6th to 10th year.

According to audiograms four of these five cases were established as

far advanced hearing losses of the mixed deafness type and one as a pure conductive deafness.

In all five cases an exceptional instantaneous postoperative success was obtained through mobilization. The postoperative audiograms show in all cases a remarkable improvement of air conduction, partly even in the pre-operatively depressed, higher frequencies, and in two of these cases a big postoperative improvement even in the bone conduction curve could be demonstrated.

In one case the improvement has lasted for two years till today.

In two cases after an initial improvement clinical refixation ensued within six months of the operation.

In two cases immediate improvement was marked, but the post-operative period of six to nine months is too short to allow for final judgement.

It is extraordinary, that in none of our five cases a family history of otosclerosis could be obtained. Not one of these cases reported an inflammatory process of the middle ear on the affected side. No case had pre-operatively a positive sign of Schwarze, as well as no tinnitus.

Discussion and Conclusions about our Five Cases

The number of five cases of juvenile otosclerosis is too small to permit definitive conclusions.

Surveying the before-mentioned cases, it is interesting to note, that our young patients had suffered some loss of hearing for an average of about six years before they consulted a doctor and then only at the request of parents or their teachers. They reported usually that in the very beginning of their unilateral deafness they did not notice that they did not hear well, and they became accustomed in the beginning to this minor disability, that even later on they were not conscious of their deafness.

The clinical beginning of their manifestation of the otosclerosis started according to the history on the average during their 6th to 10th year, but it is impossible to establish the precise beginning, because, as mentioned above, they haven't been aware of the starting disability.

Contrary to the statistical experiences of various authors, the proportion of the sexes in our cases is reversed, that is to say we found one female to four males. Perhaps this is only an accidental finding in these few cases, but there may be other ways to regard this problem too: in the opinion of a few authors the natural tendency towards the otosclerotic process is fundamentally the same in both sexes, and only afterwards, as a result of different factors and influences, it comes to the development of this process in a certain direction. One of these factors quoted is the hormonal influence, especially in the female. In cases of juveniles, however, where

the sexual endocrine influences are not yet so characterized, perhaps the preponderance of the female will not yet be so striking.

In all our cases it was easy to mobilize the stapes through the indirect way. This happened perhaps because the otosclerotic focus of the juveniles is more active and therefore of a softer consistency, than a less active and more rigid focus at a more advanced age.

Also the relatively early refixation of the footplate shows in two of the five cases—in two other cases there is as yet no certain result on account of the short postoperative period—that the operative fracture of the mobilization has most probably cut through the active otosclerotic focus, which, as known, has a great tendency towards refixation. For these reasons it would be advisable in future in juveniles to choose a by-pass operation and thus avoid the more direct approach to the active otosclerotic focus.

It is remarkable that in a relatively high percentage of our cases an affection in the high frequencies of the audiograms has been observed—by repeated audiogrammatical testings. Of these cases four had advanced mixed deafness and only one a pure conductive deafness.

The patho-anatomical basis of progressive perceptive weakness of hearing in cases of otosclerosis, which points at a weakening of the neuroperceptive endings in the Corti organ, or in the very nerve, is still to this day not completely explored. Many writers express the opinion that nerve degeneration is a secondary effect of the disease, but the exact physiology and pathology of the process which involves the nerve has not been conclusively shown.

Some authors tried to explain this degenerative process in the Corti organ or in the nerve itself by an extension of the otosclerotic bony growth to the basal turn of the cochlea and to the internal auditive duct, but the results of histological examinations show only in rare cases, that the basal coil or the meatus acusticus internus are affected through an otosclerotic process. Therefore, there is no satisfactory reason to conclude, that there is in a similar case of an otosclerotic mixed deafness a direct relation between the otosclerotic focus and the deterioration of the neuroperceptive organ.

Some other authors did assume that an eventual immobility of the endolymph, as a consequence of the closure of the oval window, may result in a degenerative process of the neuroperceptive endings, as an expression of "degeneratio ex inactivitate". But we cannot be too sure, that an immobile endolymph is resulting in an eventual degeneration of the Corti organ or of the very fibres of the hearing nerve.

An eventual other hypothesis of the deterioration of the Corti organ on a toxic basis through metabolic products, originating in the otosclerotic focus, is also not too convincing.

In this connection the findings of Bast and Anson have a certain

importance. These authors have established that there is in front of the oval window an anatomically tiny fissure, which serves as drainage of the perilymph. Should the surroundings of the fissure be affected by an otosclerotic focus, the drainage of the perilymph is cancelled. Through this fact:

1. The endocochlear pressure is increased and this hypertension automatically influences the fixation of the stapes, and

2. if this hypertensive pressure continues, it can lead to an atrophy of the neuroperceptive elements in the cochlea.

In the case of an eventual mobilization and freeing of the footplate—similar to Holmgren's ideas in the case of fenestration on the lateral canal—the result is a lowering of the intracochlear pressure and through this an improvement of the hearing capacity.

A further very impressive fact in our cases is the explicit, even though partial reversibility of the perceptive weakness, shown in the above mentioned audiograms.

In two of our cases the postoperative audiogram shows not only a very improved air conduction, but also the bone conduction has improved over the preoperative findings!—repeated audiogrammatical testings. It is remarkable, that in two of the cases with an preoperative "weakening of their cochlear reserve", an improvement was observed not only in the low and middle frequencies, but to a certain grade even in the highest ones.

The postoperative improvement of the high frequencies is very impressive. This reversibility of the decreased high frequencies is not quite understandable and shows that the eventual changes in the organ of Corti and even in the very nerve were not quite definitive!

This is not an isolated event during the past years, and cases are more and more often reported in the literature, in which the lowered level of the bone conduction in different middle-ear processes has much improved after the healing or operation of these primary causative processes. Huizing Jr. reports many similar cases and explains the postoperative improvement of the bone conduction through abolition of the stiffness in the ossicular apparatus. This improvement of hearing is then the result of freeing of the vibratory state of the endolymph.

Likewise the postoperative disappearance of the so called Carhart notch in otosclerosis belongs to this category of an improved bone conduction too.

Further it is interesting to observe, that in two of the above-mentioned cases of refixation, the aimed improvement returned again to the preoperative audiogrammatical findings. This concerns the air as well as the bone conduction. That means that the pathophysiological and functional reversibility goes parallel with the reversibility of the pathoanatomical changes on the stapedovestibular junction.

From these observations we have to deduct, that in similar cases of reversibility of a depressed cochlear function, there can be no question of a definitive state of degeneration in the neuroperceptive elements, but there must be more different other processes, which may influence the functional efficiency of the Corti organ. These other, at the present time not sufficiently elucidated processes, lead in the case of stapes fixation to a remarkable depression of the function of the neuroperceptive organ and this depression is, at least partly, in both meanings reversible. It is certain that this fact is connected closely with the footplate fixation, with the stiffness of the conductive apparatus or with the increase of the mass in this system, and as a result of that, with a diminution of the vibratory effect of the endolymphatic system. But we have to consider, that in all consequences the conductive threshold is not only the exclusive expression of an existing "cochlear reserve". There are many other, to this day, unknown influences, which are participating in the formation of the bone conduction curve!

Comments and Statistical Reports from the Literature

Our above-mentioned five cases of juvenile otosclerosis were taken out of a total of 135 cases of otosclerosis, which were operated on by us in the period from April 1956 to October 1960, i.e., out of a total of operated cases of otosclerosis, 3·7 per cent. were so-called clinical features of otosclerosis, where the symptoms were manifested during the 6th and 7th year of the juvenile.

We know from the literature that McKenzie observed among 300 clinical otosclerotic cases, eight under the age of 15 years, i.e., 2·7 per cent.

Cawthorne under	2,000 cases	1·8% under the age of 11
Wullstein under	700 cases	8·5% under the age of 16
Larsson under	262 cases	2·3% between the age of 6 to 10 and 6% between the age of 11 to 15 yrs.

From these statistical statements of different authors is to be seen, that the *clinical otosclerosis* of juveniles under 10 years is approximately 2 to 4 per cent., and with advancing age we find a progress in the percentage of occurrence of clinical otosclerosis.

In the opinion of various authors, the influence of different factors in diverse countries, as climate, racial differences, sexes, different social influences, etc. are not to be underestimated.

Guild's statistics concern cases of *histological* otosclerosis, and he proved that 9 per cent. were under 15 years of age.

This mentioned statistic of Guild, concerning the histological otosclerosis, shows clearly the difference between the occurrence of the clinical and histological otosclerosis. It is only natural if we consider that a histological process not always leads to the clinical symptoms of stapes fixation.

The *degree of manifestation* of the clinical otosclerosis is, after the observations of different authors, approximately 25 per cent. of the histological form. The clinical manifestation of otosclerosis is higher among the women than among the men, perhaps, as mentioned above, as a result of different biological and endocrine factors and influences in the organism of the woman.

According to Guild we are able to observe histologically the otosclerotic focus in the enchondral layer of the labyrinth capsule among the Whites approximately about in 5 to 10 per cent., whereas among Negroes only in 1 per cent. In the case of white women in 12·5 per cent., in cases of white men only in 6·5 per cent.

The occurrence of clinical otosclerosis among Whites is, with some certain local exceptions, between ½ to 1 per cent., whereby the relationship between men and women is here 2:3.

There is not yet much known about the *pathogenesis* of the otosclerotic process, as well as about the reasons of the activity or inactivity of an otosclerotic focus. One takes for granted endocrine factors, as lactation or gravidity, as well as different chemical, racial, social and other influences.

Dorothy Wolff believes that otosclerosis aetiologically has no specific reason at all. According to her hypothesis, the peripheral blood supply in the labyrinth capsule is damaged and the damage may be of a chemical, vasomotor, allergic or vibratory character.

Lindsay thinks that a certain influence of preceded and repeated inflammations of the middle ear may lead to appearance of an otosclerotic process in the affected ear.

By support of various statistics it is proved from case histories, that there exists a *familial disposition* to otosclerosis in about 50 to 60 per cent. In this sense we find in the statistic of

Nager	58 per cent.
Cawthorne	54 per cent.
Shambaugh, Jr.	54·4 per cent.

But Larsson asserts that from 51 per cent. of his cases with reported otosclerosis in their family, only in 79 per cent. the answer was really right, whereas in 21 per cent. of these cases there was no question of an otosclerosis, but of another kind of hard hearing.

A negative familial case history is of no great importance, because in many members of such a family there could have been present an asymptomatic histological otosclerotic process.

Concerning the *localization of otosclerosis*, we learn from the statistic of Guild, that the appearance of histological otosclerosis is in 30 per cent. unilateral and in 70 per cent. bilateral.

In the statistic of Nager the clinical otosclerosis is in 10·7 per cent. unilateral, and Larsson found in his statistic, that the clinical form is in 15 per cent. unilateral.

This difference in the localization between the clinical and histological otosclerosis is perhaps explained by the fact that the unilateral affected patients go rather less frequently to see a specialist than those who are affected bilaterally.

Diagnosis and Differential Diagnosis

Only in exceptional cases we meet with difficulties with adults concerning diagnosis and differential diagnosis. In cases, which are not quite clear we have still the possibility of an operative exploration of the middle-ear cleft. With children the problem is much more difficult, the younger they are, the more difficult it is. The parents erroneously rather believe in inattention, disobedience and incapacity to concentrate, than in difficulty in hearing of their child. The child himself is often not conscious of his affliction, especially if it is only unilateral. Perhaps there is also not sufficient possibility of comparison for the child, because in his relatively short life it had no complete chance to experience normal hearing, and it does not realize well the starting difficulty in hearing.

In all these cases the observation of parents and teachers is of great importance. McKenzie stresses that a child with a good pronunciation, sometimes even with a local accent, could not have been completely deaf from early age, and his disability must have started later, when his speech had already been developed. In such a case, e.g., we would be more inclined to think of an acquired disability than of a congenital deafness.

Diagnostically the following points are of great importance:

1. Co-operation of children under five years is extremely difficult to get, and, therefore, the diagnosis cannot be exact.

2. Parents do not usually notice a unilateral affection and mostly only at school later the teacher informs them about the difficulty in hearing. But only at the age of 10 years are we able to establish a more precise differential diagnosis. The children compensate easily for their unilateral deafness with the other good side and it becomes easy for them to lipread.

3. In the case of a bilateral affection we are inclined to trace this back rather to an otosclerotic basis, whereas in unilateral processes these mostly can be of congenital origin.

4. Most typical for otosclerosis is a conductive deafness, which by assertion of the parents has not started in early age, but progresses with the years.

5. What we learn from the case history about former injuries, operations, etc. of the ear, is important to differentiate from an otosclerotic process.

In a case of conductive deafness, where the ear drum does not show any pathological changes, we should not forget, making a differential diagnosis, the following:

1. A congenital fixation or absence of ossicles;
2. a bony fixation of the ossicles in cases of cretinism;
3. fixation of the ossicles in a case of Paget disease;
4. healed injuries of the middle-ear contents.

According to House we classify children under 12 years of age, who suffer from conductive deafness and whose ear drum is without pathological changes, into four diagnostically different groups:

1. Conductive deafness of 15–35 db., which reacts well to the usual treatment: catarrhal or allergic processes.

2. Conductive deafness of 15–35 db., which does not react to the usual therapy, but leads after the 12th year to a spontaneous regression. In these cases mostly lymphoid tissue in the neighbourhood of the eustachian tube is involved.

3. During early age a light conductive deafness, which progresses later, is typical for otosclerosis.

4. A distinct conductive deafness of 45–55 db. from early childhood with no progression: congenital changes in the middle ear.

If there are differential-diagnostical difficulties in childhood or in cases, which are not quite clear, the occurrence of otosclerosis in the family is very important.

It is very interesting to see statistical reports and notes about parallel occurrence of otosclerosis and inflammation of the middle ear: Nager finds among 1,146 otosclerotic cases in 69 per cent. the ear drum normal. Cawthorne ascertains among 2,000 otosclerotic cases in 84 per cen at. normal ear drum.

But differential-diagnostically we should not forget that a chronic adhesive process in the middle-ear cleft behind a completely normal ear drum, can also lead to a conductive deafness!

Larsson finds, that in 20 per cent. of otosclerotic cases inflammatory processes of the middle ear have preceded, and from these only half of the cases had really objective pathological changes on the ear drum. Further in half of the cases which had changes on the ear drum, the reported information about inflammatory processes of the middle ear was negative.

Prognosis

Concerning the prognosis of juvenile otosclerosis, it is important to consider the statistics of the different authors, e.g., Ruëdi, Spoendlin. Active and inactive otosclerosis appears in all stages of life, but in more advanced age the active focus dwindles. However, it is not quite clear from these statistics, how frequently the active otosclerotic focus occurs before the 10th to 12th year. Logically it is to foresee, that active foci are more predominant in early age than later.

Prognostically is to be stressed that an inactive otosclerotic process, concerning the function of the ear, is more benign, whereas an active process in this sense is less promising. That means, we have to consider, that the progression of an inactive otosclerotic process is less dangerous for the functional injury of the ear, and that goes as well for the fixation of the vestibulo-stapedial junction, as for an eventual injury of the neurosensorial apparatus. In respect of an intended operation the more active focus is prognostically less to its advantage.

In our cases we could observe, that out of five cases two were refixed after performed mobilization relatively, after a short period. This fact is perhaps in accordance with the above-mentioned conclusions and with our presumption, that more active processes are possibly present in juveniles and these tend to refixation. We can conclude perhaps, that in juvenile otosclerosis "not enough time" has passed, that an active process has changed into a less active state.

Further, we wish to stress here, that a positive Schwarze sign prognostically is less advantageous, because this shows a diffuse, red and active otosclerotic process.

According to Brown-Farrior stapes surgery is rarely of a lasting value in diffuse, red and vascular otosclerosis, which extends over the entire promontory. White, avascular otosclerosis is most favourable for lasting results in mobilization surgery, while in red, vascular forms mobilization can be accomplished, but with almost uniform refixation.

Therapy

Treatment in juvenile otosclerosis is logically, as in all other cases of this disease, an operation.

In course of an eventual diagnostical exploration of the middle ear in a case which preoperatively has not been quite clear, we have to try a mobilization. Should this mobilization not be of a lasting result, we have to decide between an eventual remobilization by by-passing, or a fenestration of the lateral canal. In a case of a recognized active otosclerosis we would prefer in every case a by-pass method, fearing that a new reankylosis of the footplate in the active focus may occur.

Conclusions

From a total of 135 operated otosclerotic cases we observed in 3·7 per cent. cases of so-called juvenile otosclerosis.

The clinical signs of their hearing disability started on an average at the age of 6 to 10 years.

They attended a specialist only at the average age of 12 to 16 years. There have been four males and one female under our observation. We observed a relatively high percentage of mixed deafness among our cases.

The weakness of the neuroperceptive part of this mixed deafness improved remarkably after a successfully performed mobilization.

In two cases a big improvement postoperatively in bone conduction has been observed too.

In two cases, after a relatively short postoperative period, the achieved improvement returned to the preoperative findings. We assume here a postoperative refixation in the active otosclerotic focus.

On the basis of the postoperatively observed reversibility of the neuroperceptive weakness, we assume that different other influences are participating on the creation of the bone conduction curve, and not only the so-called "cochlear reserve" is here decisive! According to Huizing Jr., one of these influencing factors is the rigidity and the increased mass in the conductive apparatus of the middle ear. As a result of this fact the vibratory capacity of the endolymph is decreased.

There is assumed, that an active otosclerotic focus occurs more frequently in juveniles, than later on in the advanced age, and therefore a reankylosis of the footplate in such an active otosclerotic focus in juveniles is more probable and more often occurs.

Summary

Different types of juvenile conductive deafness have been reported.

Five cases of juvenile otosclerosis in our practice have been shown and discussed.

Some very interesting accompanying symptoms have been observed as i.e., postoperative improvement of hearing not only through air, but also through bone, postoperative reversibility of a preoperative neurperceptive weakness, etc.

Statistical details about histological and clinical otosclerosis from the literature, occurrence, pathogenesis, diagnosis and differential diagnosis, prognosis and therapy of juvenile otosclerosis have been discussed.

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DRAINAGE OPERATION IN CASES OF THE HYDROPS OF THE LABYRINTH AND ITS INFLUENCE ON HEARING*

By B. FEMENIĆ, M.D. (Zagreb)

At the annual meeting of the Austrian otologists in Salzburg, in 1952, I was able to demonstrate for the first time a case of the hydrops of the labyrinth in which permanent drainage of the endolymphatic system into the perilymphatic space was achieved. The result of the intervention was very satisfactory, as tinnitus and vertigo were both eliminated while the cochlear function was preserved.

Two years later, at the Fifth Congress of Yugoslav Otologists in Zagreb, I reported on three more cases treated by the same method. The results were again satisfactory and the operation was accepted as a routine procedure for surgical treatment of the hydrops of the labyrinth at our Clinic.

The method applied may be briefly described as follows:

Lempert's fenestration is first performed, with this essential difference that the fenestra is created at the farthest possible point of the ampullary end of the semi-circular canal, instead of in the dome of the vestibule (Fig. 1). Subsequently a stopple corresponding exactly to the fenestra is made of cartilage taken from the auricle. The membranous semicircular canal is then cut through with a sharp knife against that edge of the fenestra which lies opposite its ampullary end. The part of the membranous semicircular canal which remains afloat in the perilymph is bent over the edge of the fenestra into the trepanation cavity (Fig. 2). Care should be taken that this is done without exerting strain on the labyrinth. Finally, the fenestra is closed with the prepared stopple, thus compressing the membranous semicircular canal and at the same time fixing it into position (Fig. 3).

By this procedure we are convinced to have obtained permanent drainage of the endolymphatic system into the perilymphatic space because it leaves intact the connective tissue ligaments supporting the membranous canal afloat in the bony canal, which prevent the opened part of the membranous labyrinth from collapsing and closing (Fig. 4).

*This paper was read at the "Greek-Yugoslav Congress of Otology, Neurology and Ophthalmology"—Belgrade (Yugoslavia) September 5th. It was dedicated to my chief and teacher Professor Dr Branimir Gušić in honour of his 60th birthday.

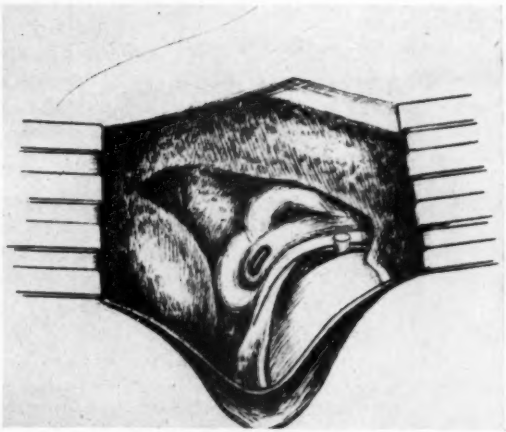

FIG. 1.

Creating of a fenestra at the farthest point of the ampullary end of the semicircular canal.

FIG. 2.

Cutting through the membranous semicircular canal against the edge of the fenestra lying opposite its ampullary end and bending over of a part of the membranous semicircular canal into the trepanation cavity.

FIG. 3.
Closing the fenestra with a cartilage stopple.

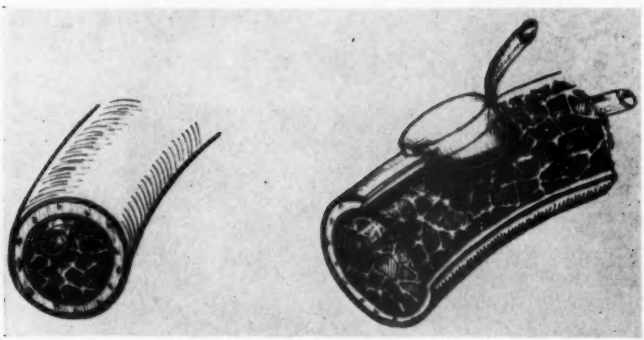

FIG. 4.
The preserved connective tissue ligaments securing the permanence of the drainage opening.

Among the results obtained by this method one fact attracted my attention. No matter whether pre-operative hearing was better for air conduction or bone conduction, the audiograms taken after the operation showed always the bone curve to be on a higher level than the air curve. My explanation was that this phenomenon was due to the interruption of the ossicular chain. So I was led to try and modify my procedure so as to leave the ossicular chain intact. This aim was achieved by the following method.

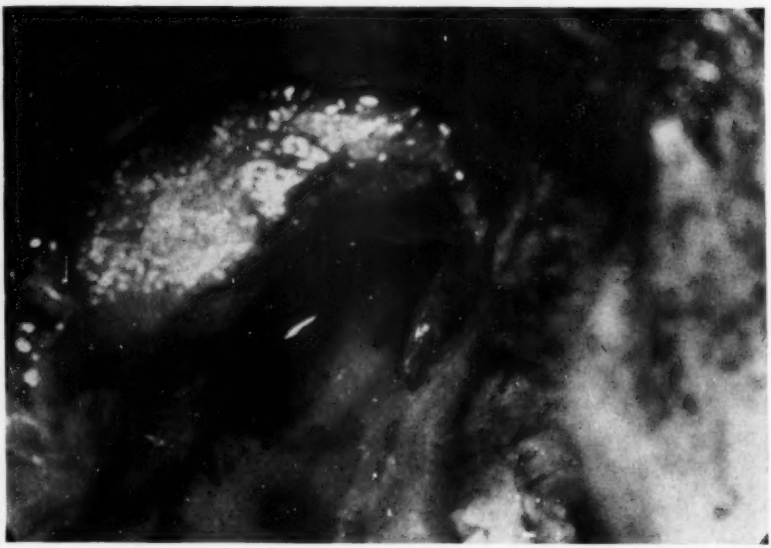

FIG. 5.
The fenestra on the exposed semicircular bony canal.

The mastoid is first explored through the endaural approach. Subsequently, trepanation of the mastoid bone is performed in such a way that the thinned end of the superior osseous meatal wall bridging across the notch of Rivinus is preserved, whereas the long process of the incus and the bony semicircular canal come into view. The exposure of the canal thus obtained is wide enough to permit the creation of a fenestra, which is done and the operation terminated along the same lines as in the original method (Figs. 5, 6 and 7).

Unfortunately, this modified version leaving intact the ossicular chain failed to confirm my hypothesis as the hearing loss was again without exception that of the conductive type (Figs. 8 and 9). No other explanation of this phenomenon is submitted at this moment since it would be necessarily based on speculation.

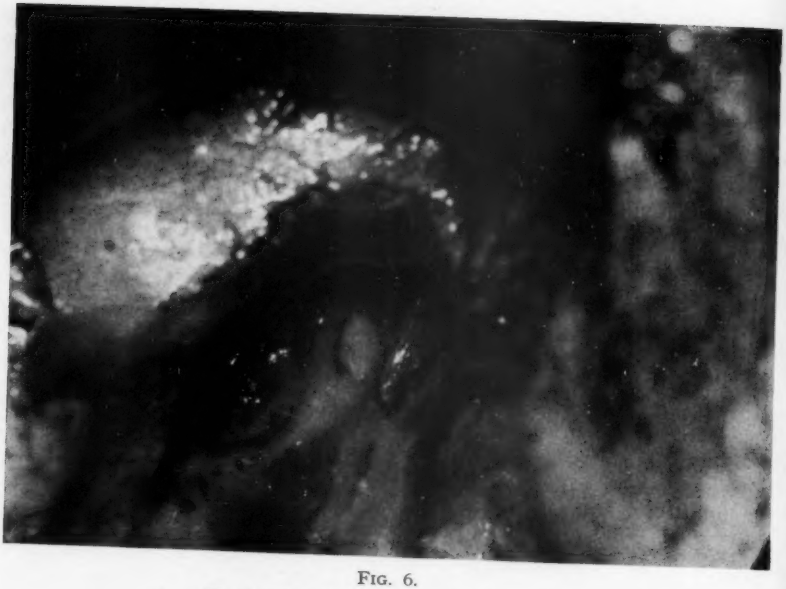

FIG. 6.
The fenestra closed by a cartilage stopple.

FIG. 7.
The fenestrated semicircular bony canal covered with the tympano-meatal plastic flap.

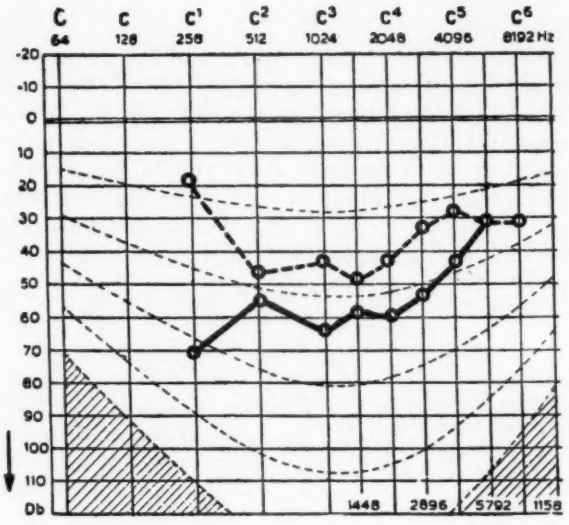

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FIG. 8.

Pre-operation audiogram. ————air conduction
..... bone conduction.

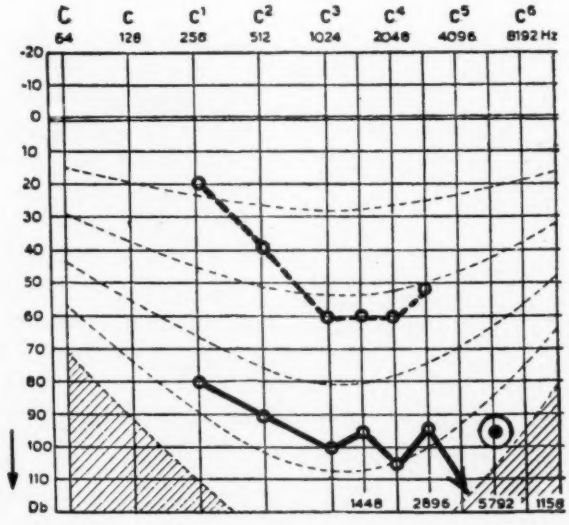

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FIG. 9.

Post-operation audiogram. ————air conduction
..... bone conduction.

However, the operation brought to light still another puzzling fact. It showed incontrovertibly that, contrary to what has been asserted so far, even in cases of the hydrops of the labyrinth the function of the cochlea is preserved, although in a diminished degree, after the interruption of a lateral membranous semicircular canal. The preserved hearing may seem of little practical use to patients whose other ear is normal, but we must never forget that the apparently normal ear may become affected at any time. If this happens the patient will be grateful for having had his hearing preserved in the operated ear.

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"MASTOID OSTEOPLASTY" USING AUTOGENOUS CANCELLOUS BONE

PROGRESS REPORT AND MODIFIED TECHNIQUE

By A. SCHILLER (Cape Town)

THIS new surgical procedure was first described by Schiller and Singer in 1960. It utilizes autogenous cancellous bone strips to obliterate old mastoid cavities and large cavities freshly constructed in performing modified and radical mastoidectomies, fenestrations and tympanoplasties.

The strips are removed from an iliac crest and inserted into the cavity to reconstruct the exenterated petromastoid and restore the normal contour of the external auditory canal. Specific skin flaps are constructed to nourish the grafts, thus providing a healthy epithelial covering to the reconstructed lateral surface of the mastoid and the posterior bony wall of the canal.

The procedure was specifically designed to obviate all the following undesirable features frequently associated with the cavity.

1. Prolonged after-care.

2. Aural discharge.

Patients left with a discharging ear following fenestration or tympanoplasty are invariably dissatisfied, particularly if there has been no improvement in their hearing.

3. Hearing-aid problems, as follows:

(*a*) The acoustic qualities of the external auditory canal are altered when a large mastoid cavity is constructed, so that if a hearing-aid is required the patients discover that, although the sound is amplified, it is distorted.

(*b*) It is seldom satisfactory to construct a mould for a hearing-aid insert to an ear with a large mastoid cavity and an enlarged meatal opening.

(*c*) Stenosis of the meatus is a complication not often encountered but, when it does occur, it becomes difficult to carry out aural toilet. It is usually impossible to fit a hearing-aid insert, should that be required.

(*d*) In the presence of a discharging cavity, it is undesirable to fit a hearing-aid insert.

Previous Methods used to Promote Healing of the Cavity

The variety of methods hitherto used to enable the cavity to heal is indicative of their inadequacy. These are:

1. Skin Grafts

The cavity is usually lined with Thiersch or Woolf grafts, either as primary or secondary grafts. This method promotes rapid healing, but in a large number of cases the skin breaks down and the cavity becomes troublesome.

2. Pedicle Flap (Beales and Hynes)

This flap lines the cavity adequately but it does not obviate the hearing-aid problems.

3. Muscle Flaps

(*a*) *Temporalis muscle flap*. Rambo uses a flap of temporal muscle to reconstitute the tympanum and to line the mastoid cavity. This method is only applicable to tympanoplasties—type 5.

(*b*) *Sternomastoid muscle flap*. Guilford constructs a pedicle flap from the sternomastoid muscle and reflects it upwards to line the cavity and to reduce its size. Although this may be satisfactory, it must be remembered that, when active muscle is reduced to a passive state, the muscle fibres tend to atrophy and eventually become replaced by fibrous tissue. Should this occur, the flap of muscle will become reduced in bulk and the cavity will assume its orginal proportions.

4. Diced Cartilage and Mastoid Bone Chips

Guilford uses either diced heterogenous or homogenous cartilage to fill in large cells that become exposed during exenteration of the mastoid bone in fenestration operations. Unfortunately, this excellent method of repair has been found to be unsatisfactory when used to obliterate large cavities in bone and to restore contour, since cartilage tends to become replaced by fibrous tissue and it is not suitable for grafting in the presence of infection.

Prior to the use of cartilage for this purpose, Guilford used bone chips from the mastoid process to fill the defects. He discontinued this method because he found that the bone chips remained inert and acted as foreign bodies.

The New Concept

It was considered that if the mastoid cavity could be completely obliterated it should be possible to obviate all the disadvantages associated with it. In consequence, a one-stage procedure was devised which aimed

at both total reconstruction of the exenterated petromastoid including the posterior bony wall of the external auditory canal and provision of a healthy epithelial lining to the reconstituted canal. It could be utilized in modified and radical mastoidectomies, tympanoplasties types 3-5, fenestrations and obliteration of old mastoid cavities, even in the presence of infection.

Autogenous cancellous bone strips obtained from an iliac crest were selected as the most suitable grafting tissue for this purpose. The cancellous strips are nourished by the tissue fluids for 72 hours before they are revascularized; moreover it is known from grafting procedures on the mandible and elsewhere that the cancellous strip grafts will survive, even if moderate infection is present.

Specific skin flaps were constructed from the soft tissues which cover the posterior surface of the auricle and postauricular area, and also from the skin lining of the membranous and bony portion of the external auditory canal. These flaps served to provide additional nourishment to the grafts and to furnish the reformed external auditory canal with a healthy viable epithelial lining.

Observations

The procedure was first performed in October 1959. To date 23 cases have been treated by this method (Table I). In this article it is intended to report on the progress of these cases in order to indicate and discuss the behaviour of the grafts and to describe the present technique, which has been modified to simplify the procedure.

TABLE I.

TWENTY-THREE CASES WITH BONE GRAFTING OF THE MASTOID CAVITY

Case No.	Operation and Date	Canal Dry and Epithelized Post-op	Contour of Canal	Mastoid Cavity	Reduction in Bulk	Sequestration	Radiographic Integration
1	Tymp. 3 + M.O. 2.10.59	17 days	Normal	Obliterated	Yes. Canal-widened 6th month	Nil	4 months
2	Fen. + M.O. 14.10.59	3-4 weeks	Normal Outer ⅓	Obliterated	Nil	Nil	4 months
3	O.M.C. + M.O. 12.11.59	3-4 weeks	Wide	Obliterated	Nil	Nil	4½ months
4	Tymp. + M.O. 28.11.59	3–4 weeks	Normal	Obliterated	Nil	Nil	4 months
5	Tymp. + M.O. 12.11.59	3–4 weeks	Normal	Obliterated	Nil	Nil	3½ months
6	Tymp. + M.O. 23.1.60	3–4 weeks	Normal	Obliterated	Nil	Nil	4½ months
7	Tymp. + M.O. 9.2.60	3–4 weeks	Patient could not be traced after 5th week				
8	Fen. + M.O. 18.2.60	3–4 weeks	Normal Outer ⅓	Obliterated	Nil	Nil	4 months

Case No.	Operation and Date	Canal Dry and Epithelized Post-op.	Contour of Canal	Mastoid Cavity	Reduction in Bulk	Sequestration	Radiographic Integration
9	Tymp. + M.O. 24.2.60	3–4 weeks	Normal	Obliterated	Nil	Nil	4–5 months
10	Tymp. + M.O. 7.3.60	3–4 weeks	Normal	Obliterated	Yes. Canal-widened 6th month	Nil	4–5 months
11	O.M.C. + M.O. 10.3.60	New flap made 5th week. Dry 8th week	Wide	Obliterated	Nil	Nil	Integrated 7th month
12	Tymp. + M.O. 23.3.60	3–4 weeks	Normal	Obliterated	Nil	Nil	4–5 months
13	Tymp. + M.O. 10.5.60	3–4 weeks	Normal	Obliterated	Nil	Nil	5–6 months
14	Tymp. + M.O. 24.5.60	3–4 weeks	Normal	Obliterated	Nil	Nil	5–6 months
15	O.M.C. + M.O. 29.5.60	Fungus infection 3rd week. Dry 4th week	Wide	Obliterated	Nil	One graft visible 2nd month. Remainder becoming revascularized	
16	Tymp. + M.O. 19.6.60	3–4 weeks	Normal	Obliterated	Nil	Nil	Grafts have lost their outline and are becoming revascularized
17	Tymp. + M.O. 19.6.60	3–4 weeks	Normal	Obliterated	Nil	Nil	ditto
18	Tymp. + M.O. 15.7.60	3 weeks	Normal	Obliterated	Nil	Nil	ditto
19	Fen. + M.O. 29.7.60	3 weeks	Normal Outer ⅓	Obliterated	Nil	Nil	ditto
20	Tymp. + M.O. 3.8.60	3–4 weeks	Normal	Obliterated	Nil	Nil	ditto
21	O.M.C. + M.O. 7.8.60. Scholtz	Moist	Wide	Partially obliterated	Nil	Yes. 3 strips near to Tegmen	
22	Tymp. + M.O. 14.9.60	3–4 weeks	Normal	Obliterated	Nil	Nil	Not taken
23	Tymp + M.O. 28.9.60	3 weeks	Normal	Obliterated	Nil	Nil	Not taken

Tymp. = Tympanoplasty M.O. = Mastoid Osteoplasty O.M.C. = Old Mastoid Cavity
Fen. = Fenestration M.C. = Mastoid Cavity

Case XV—Fungus infection in canal occurred during third week.
Responded to Nystatin ointment locally.
Single Sequestrum visible radiologically due to Aseptic Necrosis.

Case XXI—Postauricular Flap cut too short.
Flap failed to cover the grafts near Tegmen. These sequestrated.

Total number of cases done—23.
Clinically successful —22 cases.
Radiographic integration —Complete —13 cases.
—Incomplete — 9 cases. (Revascularized. Incorporation is proceeding but is not complete.)
Clinical and radiographic failures — 1 (Case XXI) due to technical error.

Weekly clinical observations, monthly radiographic studies, and exploration of three of the cases during the 5th post-operative week, have all shed considerable light on the changes that take place in the grafted area. It will be shown that autogenous cancellous bone strips,

grafted to the mastoid bone in order to restore its contour, undergo similar changes to those reported by surgeons who have used this medium to repair fractures of long bones, or to restore the contour of the dorsum of the nose.

A. Behaviour of Autogenous Cancellous Bone Grafts in Relation to Long Bones and Nasal Bones

When transplanted the grafts undergo one of five changes. The ultimate fate varies with conditions prevailing in the grafted area.

1. Integration with the Host Bone

If adequately nourished, the grafts become replaced by new bone by a process of resorption, revascularization and apposition. This takes place even if moderate infection is present in the grafted site.

2. Sequestration

This may occur if the grafts are inadequately nourished (aseptic necrosis) or become grossly infected.

3. Replacement by Fibrous Tissue

It has been revealed that when autogenous cancellous bone is used in the form of slivers or chips, these tend to become replaced by fibrous tissue. It is contended that, in this form, the bone acts like a foreign body and stimulates an invasive fibroblastic response from the surrounding tissues.

4. Absorption

Allen and Goldman reported that in many instances the autogenous cancellous bone strips (used to restore the contour of the nasal dorsum) become absorbed after an interval of 9-12 months. No explanation for this has been given.

5. Reduction in Bulk

Peer reported that where autogenous cancellous bone grafts are utilized to fill in a gap and restore bone contour in areas not subjected to stress and strain, the grafts become replaced by new bone and integrate with the host bone. They tend, however, to diminish in bulk.

B. Behaviour of Autogenous Cancellous Bone Strips Grafted into the Mastoid Cavity

Complete obliteration of the mastoid cavity occurred in all cases. The following are illustrative cases:

1. *Resorption and Revascularization of the Grafts*

Male aged 9 years (Case 8). Operation: Tympanoplasty and mastoid osteoplasty.

In the fifth post-operative week a bulging inwards of the reconstructed posterior bony canal wall was noted. Faulty clinical judgement led me to believe that the grafts were sequestrating. I decided to explore. A post auricular incision was made and the soft tissues were carefully dissected forwards to expose the grafted area.

This revealed a fascinating picture. The outermost discernible grafts had lost their dense concellous structure. They appeared as red gelatinous rods embedded in a red, finely-reticulated jelly-like matrix, formed by the new blood vessels and connective tissue strands extending into and invading the grafts from the host bone. The rods were narrower and shorter than the original cancellous bone strips inserted. As it was clearly evident that resorption and revascularization of the grafts had taken place, I resutured the wound without disturbing the grafted area.

I regret I did not remove a section for histological examination.

Subsequent progress was uneventful and the grafts eventually became replaced by new bone radiologically indistinguishable from the host bone.

2. *Sequestration*

Male aged 40 years (Case 19). Operation: Mastoid osteoplasty to a stenosed and infected radical cavity.

When the patient was examined three weeks post-operatively, the auditory canal was found to be heavily infected with fungus. This was meticulously removed and intensive local treatment using nystatin ointment was instituted. Although the response was rapid and the canal appeared to be clear after treatment over a period of five days, it was not apparent whether the grafts had become infected. The patient was observed for a further four weeks and then sent for radiographic studies. A single area of density surrounded by a clear zone was clearly visible in the vicinity of the facial ridge. The other grafts had lost their outline and the area representing them appeared less dense than the host bone, indicating that the grafts were revascularized.

The advisability of exploring the area to discover whether the sequestration was the result of infection or aseptic necrosis was discussed with the radiologist. It was eventually decided that, since there was no clinical evidence to indicate infection being the cause of the sequestration, the only logical assumption we could entertain would be one establishing the existence of aseptic necrosis of that strip of bone. In such an event the sequestrum usually remains inert or becomes absorbed. It is, therefore, unnecessary to remove this surgically. Observation over a period of a further eight weeks inclines me to believe that the assumption

that it is an aseptic necrosis is correct. The cavity remains obliterated and the canal dry.

3. Reduction in Bulk

In two patients, aged 7 and 11 years, a gradual increase in the width of the bony portion of the external auditory canal was noted between the 6th and 7th month post-operatively. As radiographic studies indicated that the grafts had become replaced by new bone and had completely integrated with the host bone, the logical deduction is that during the process of becoming consolidated, the new bone loses some of its bulk.

As this change was not noticed in the older patients, the reduction in bulk cannot be explained entirely according to Peer's theory, i.e. being due to the absence of stress and strain in the grafted area. It seems reasonable to postulate that the changes are, in some way, related to the immaturity of the host bone and the autogenous transplants.

4. Graft acting as Scaffold

Male aged 6 years. Mastoid osteoplasty to obliterate a large old radical cavity infected with cholesteatoma.

Using the conventional postauricular approach the cavity was exposed and prepared and reconstructed with autogenous cancellous bone strips. These were covered with two free full-thickness skin grafts taken from a thigh. Cleaning the cavity on the fourteenth day, it was found that the skin grafts had sloughed. The outer strips of bone were exposed.

As there was no visible evidence of revascularization of the strips by the end of the 4th week a section of a strip was removed for histological examination. This was reported on as "dead bone devoid of osteogenic tissue".

In spite of this report it was considered possible that those grafts in contact with the host bone were being revascularized and, if adequate nourishment and cover could be provided, the exposed strips of bone too might become revascularized.

Five weeks post-operatively a postauricular flap (Beales and Hynes) was constructed to cover the grafts. Subsequent progress was gratifying. For the following six months the ear was examined each week and radiographic studies were taken each month. Clinically, the mastoid cavity remained obliterated and the canal remained completely epithelialized and dry.

Early radiographs had revealed that the grafted area became increasingly rarefied, suggesting that the grafts had either become replaced by fibrous tissue or were being revascularized.

In subsequent radiographs, the area was observed to become denser indicating that new bone was being deposited.

This case supports those who subscribe to the theory that the cancellous strips need not survive as living bone but can act purely as a scaffolding for invasion by new blood vessels and connective tissue from surrounding tissues.

Procedure

A. Pre-Operative Preparation

1. Radiographs of Mastoids

Radiographs indicate the extent of sclerosis. If there is dense sclerosis, bone grafting may not be indicated. The cavity is usually small and not troublesome in such cases.

2. Bacteriological Study and Antibiotics

Where the middle ear or external auditory canal is infected, aural swabs are required for culture and sensitivity tests. The appropriate antibiotic is administered orally or intramuscularly. It is also instilled locally for 48 hours before the operation. Where no infection is present, a broad spectrum antibiotic such as tetracycline phosphate is used for 48 hours pre-operatively.

3. Skin Preparations

The mastoid and the ipsilateral or contralateral hip and thigh are prepared in the ordinary manner.

B. Technique

In order to simplify the procedure several of the original skin flaps have been modified and insertion of the cancellous bone into the cavity is confined to the reconstruction of the petromastoid and posterior bony wall of the canal. No attempt is made to reconstruct the outer attic wall.

Stage 1. Approach

It is better to use the postauricular approach than the endaural since it is essential to preserve all the skin and soft tissues of the meatal opening and external auditory canal. These are necessary for the formation of the skin flaps.

Stage 2. Formation of the Skin Flaps

Specific skin flaps have been designed to meet the requirements of the various operations. They are: (*a*) posterior hinge flap, (*b*) auricular flap, (*c*) fenestration flap, (*d*) modified Korner's flap, (*e*) postauricular conversion flap and (*f*) postauricular rotation flap. Of these, (*a*) and (*b*) are used in modified and radical mastoidectomies and tympanoplasties type

3-5, (*b*), (*c*) and (*d*) are used in fenestrations and (*e*) and (*f*) are used in reconstruction of old mastoid cavities.

To clarify the construction of these flaps it is necessary to describe them in relationship to each operation. This will be done later.

Stage 3. Exenteration of Bone

The methods of exenteration of the mastoid bone have been well described by many authors. The only variation in this procedure is to avoid exposing the dural plate and the thin bone covering the lateral sinus. This bone, being compact, has a poor blood supply and therefore inadequate regenerative powers.

Stage 4. Cancellous Strip Grafts

FIG. 1.

FIG. 2.

FIG. 3.

The ipsilateral or contralateral iliac crest is approached *viâ* a 3-inch incision extending posteriorly and slightly laterally from the anterior superior iliac spine. The iliac crest is then denuded of periosteum for 2-2½ inches. Within this limit, one horizontal cut and two vertical cuts (½-inch long) are made with an osteotome through the outer cortical layer of cancellous bone and a "lid" of the crest is swung medially (Fig. 1). Now two further vertical cuts (2½-3 inches) are made with an osteotome within this limit through the outer cortical layer only, and this flap of cortical bone, which is hinged below, is pushed laterally, exposing the middle cancellous layer (Fig. 2). Then two or more strips of cancellous bone, measuring 1½ inches by ⅛ of an inch, are removed with a sharp osteotome and are later cut and shaped into further strips as required for the cavity (Fig. 3). This is easily done with a small, sharp, slightly angled pair of bone-cutters. These strips are placed in a bowl containing penicillin solution, until required. The "lid" and the lateral cortical flap are replaced and the wound is carefully closed in layers after all bleeding has been controlled.

Stage 5. Reconstruction of Exenterated Petromastoid and Posterior Bony Wall of the Canal utilizing the bone strips, as shown in Figs. (4*a*) and 4(*b*).

It will be noted in Figs 4(*a*) and 4(*b*) that the posterior bony wall of the canal is lengthened as it meets the tegmen by extending across the space previously occupied by the aditus.

When introducing the grafts it is desirable:

1. To avoid crushing or pulping the strips—this destroys the osteogenic cells and the excellent natural scaffolding for invasion of new bone-forming elements from the host bone;

2. To place the strips so that the largest possible surface comes into contact with the host bone;

3. To avoid inserting too many strips of bone, because if an excess is used, too narrow an external auditory canal is created, and the skin flaps do not entirely cover the exposed surfaces of the grafts;

4. To place the fenestration flap or the tympanic free-skin graft in position before inserting the bone strips.

Stage 6. Replacing the Skin Flaps

The flaps are carefully replaced to cover all the exposed surfaces of the graft. Free-skin grafts used to cover any of the bone strips do not survive. The external auditory canal is then loosely packed with absorbable gelatin sponge and paraffin-gauze strips.

Finally, the postauricular surface can be covered with free split-thickness grafts taken from the thigh or inner aspect of the arm.

Post-Operative Treatment

Antiobiotic therapy is continued until the sutures are removed on the 7th post-operative day. The meatal gauze pack is removed and the liquefied or semi-liquefied absorbable gelatin sponge is evacuated by gentle sucking and/or mopping. A general anæsthetic is not required for this. Afterwards 1·5 per cent hydrocortisone acetate with neomycin sulfate drops are instilled into the meatus twice a day. On the 12th post-operative day (with children), or 14th (with adults), sutures at the iliac crest are removed and the patient can then commence walking. Radiological examinations are made at monthly intervals until it is evident that the graft has become an integral part of the mastoid bone.

Contra-Indications

In the following conditions the grafting of the mastoid cavity with cancellous bone should not be undertaken:

1. Middle-ear infection, associated with complications.

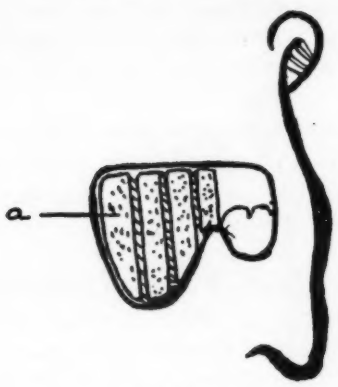


FIG. 4(*a*).

Superimposed strips of cancellous bone *a* are inserted in a lengthwise manner to extend between the tegmen and the lower margin of the cavity until the entire petromastoid and posterior bony canal is reconstructed.

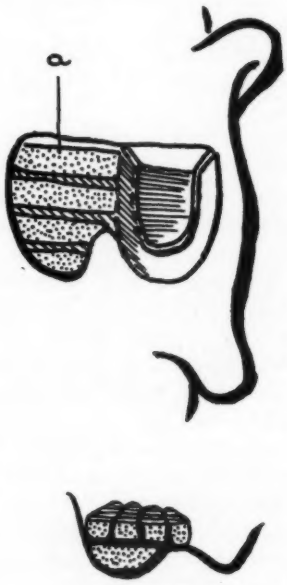


FIG. 4(*b*).

If the floor of the cavity meets the tegmen at an acute angle, it is often simpler to insert the first layer of strips *a* horizontally in order to level off the floor of the cavity. Subsequent strips can then easily be inserted lengthwise to extend between the tegmen and the lower margin of the cavity.

2. Dense, sclerotic mastoid bone. In these cases, the eventual mastoid cavity is usually small and not troublesome. Moreover, the avascular sclerotic bone is a poor, unsuitable bed for the cancellous bone strips.

Mastoid Osteoplasty in Relation to the Operations

In order to clarify the construction and placement of the various skin flaps and to follow the sequence of the procedure, the operations will be described as separate entities.

A. Modified or Radical Mastoidectomies or Tympanoplasties

The usual postauricular incision is made down to temporalis muscle and bone and the soft tissues are dissected forwards to expose the lateral surface of the mastoid and the outer attic wall. The external auditory canal is entered by making a transverse incision through the skin of the membranous posterior canal wall. This incision is extended circumferentially from 12 o'clock to 6 o'clock to divide the skin of the entire posterior and superior meatal walls and also part of the anterior meatal wall. This enables the auricle to be reflected forwards and the canal and tympanum to be visualized.

The auricle and its soft tissues, which include part of the post membranous canal wall and part of the postauricular soft tissues, constitute the auricular flap.

The posterior hinge flap (Fig. 5) is now constructed.

Having constructed the flaps, the bone is exenterated to complete the modified or radical mastoidectomy and, if desired, a tympanoplasty is performed. The full-thickness skin graft for the tympanoplasty is obtained from the posterior surface of the auricle.

It is essential to remove every visible shred of diseased tissue from the cavity, before inserting the bone grafts. The cancellous bone is removed from the iliac crest during this stage, if done by an assistant, or after this stage, if done by the surgeon. The large strips of bone are cut into smaller strips $1\frac{1}{2}$ inches long by $\frac{1}{8}$ of an inch wide and these are soaked in a penicillin solution until ready for use.

To reconstruct the petromastoid and posterior bony wall of the canal the strips are cut to the desired lengths and inserted into the cavity in the manner indicated in Fig. 4(*a*) or 4(*b*).

When this has been completed the posterior hinge flap is unfurled and positioned to cover those grafts forming the reconstituted posterior canal wall extending as far as the tegmen (Fig. 6).

The canal is then filled with absorbable gelatin sponge soaked in penicillin solution and the auricle is released. When positioning the auricle for resuturing it will be observed that its soft tissues cover those bone strips forming the lateral surface of the mastoid.

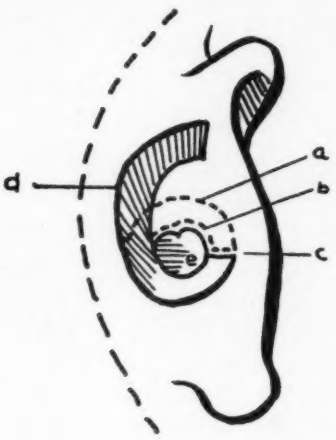


FIG. 5.

Within the confines of the circumferential incision *a* the skin lining the bony canal wall is carefully separated down to near the tympanic sulcus; where it is incised *b* circumferentially. The large flap of skin is divided *c* at its anterior attachment to form a long posterior hinge flap *d*. This flap is rolled upon itself to rest on the floor of the canal where it is protected from injury during the process of exenterating the mastoid bone. The drum or promontory is marked *e*.

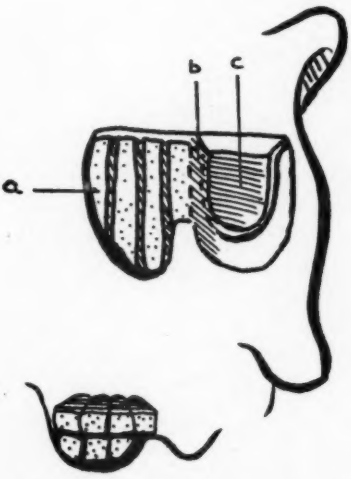


FIG. 6.

Bone strips *a in situ*. The posterior hinge flap *b* unfurled and positioned to cover the grafts of the reformed posterior bony canal wall extending to the tegmen. The full-thickness skin graft *c* forms the drum and extends onto the medial and the superior attic walls.

Finally, the incision is sutured, a short paraffin-gauze plug is inserted into the meatal opening and the raw area on the posterior surface of the auricle is covered with a free-skin graft taken from a thigh.

B. Fenestration Operation

Using the postauricular approach the fenestration operation is carried out in the usual manner. The cancellous bone strips are best taken from the iliac crest by an assistant at the time that the auricular and fenestration flaps are being constructed. If the surgeon is to do this himself, it is best done after the fenestration has been completed.

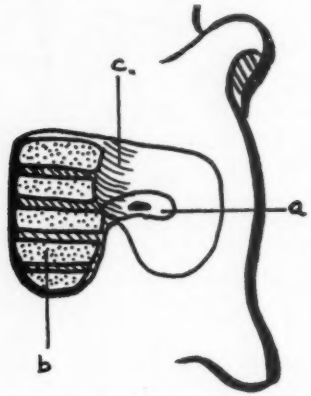


FIG. 7.
Lateral semicircular canal *a* with fenestra. The strips of cancellous bone *b* are inserted but do not encroach on the lateral semicircular canal *a*. The fenestration flap is marked *c*.

The strips are trimmed and inserted into the cavity in the manner indicated in Figs. 4(*a*) or 4(*b*). The entire mastoid cavity and part of the posterior bony meatal wall is reconstructed but care is taken not to encroach on the lateral semicircular canal. That portion of the fenestration flap immediately above and posterior to the semicircular canal is gently elevated and positioned to cover the adjacent bone strips.

In order to cover the remaining exposed strips forming the posterior wall of the canal a modified Korner's flap is constructed, as shown in Figs. 8(*a*) and 8(*b*).

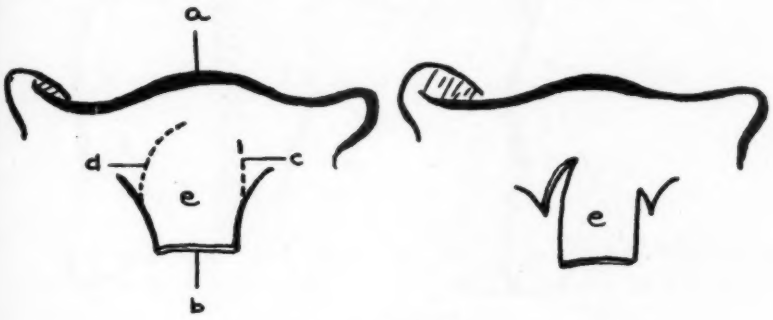
When repositioning the auricle to resuture the postauricular incision it will be noted that the modified Korner's flap hangs down in a curtain-like manner to cover the grafts forming the posterior bony canal wall. The postauricular skin with its soft tissues covers the grafts forming the lateral surface of the mastoid.

Finally, the postauricular incision is resutured and the canal is packed with a paraffin-gauze strip.

C. Mastoid Osteoplasty to an Old Mastoid Cavity

As the skin lining of the canal is deficient, flaps cannot be constructed in the usual manner. It is essential to plan a flap which will serve to cover and nourish the grafts forming the reconstructed lateral surface of the mastoid and posterior canal wall. Two flaps have been designed for this purpose. Either of these is suitable.

(*a*) *Postauricular Conversion Flap:* This flap can be used in all cases. It incorporates the principle of the Beales and Hynes flap but it is so designed that, if it is found to be inadequate in length or mobility, it can easily be converted into a rotation flap.



FIGS. 8(*a*) and (*b*).

a represents auricle. *b* is the transected end of the posterior membranous canal. A vertical incision *c* is made through the most inferior part of the posterior membranous wall and through its most superior part another semilunar incision *d* is made. The flap of skin *e* thus formed can be further mobilized and lengthened by removing a wedge of the subcutaneous tissues binding it to the auricular cartilage.

(*b*) *Postauricular Rotation Flap:* This flap can be constructed—(i) primarily as a rotation flap in all cases or, if so desired, only in those cases with small auricles; or (ii) secondarily, from the postauricular conversion flap.

1. Using the Postauricular Conversion Flap

The first step is to plan and construct the flap. This is illustrated in Figs. 9(*a*) and 9(*b*).

Preparing the Mastoid Cavity and Taking of the Bone Strips

Having constructed flap *e* (Fig. 9), the exposed deep fascia on the lateral surface of the mastoid is incised down to bone approximately ¼ of an inch behind the posterior margin of the old mastoid cavity and is continued along its superior border. The soft tissues are dissected well forwards and then transected to expose the entire mastoid cavity and tympanum.

This also enables the auricle to be reflected forwards. All diseased bone and tissue is meticulously removed from the cavity and tympanum using high-power magnification, and the bone forming the base of the cavity is freshened. It is usually found expedient for an assistant to take the iliac grafts during this stage.

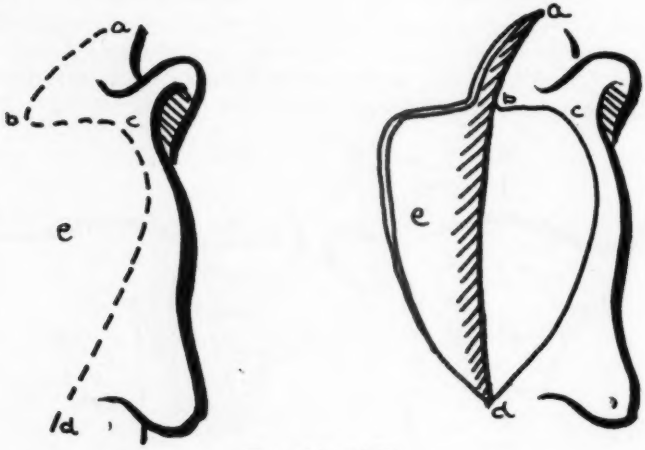


FIG. 9(*a*) and (*b*).

The line of incision *a-b-c-d* is outlined with Bonny blue solution. The skin on the posterior surface of the auricle is incised along line *b-c-d*. Point *b* is approximately ½ an inch behind the retroauricular sulcus and point *d* is at the sulcus, almost at the level of the mastoid tip. A large flap of skin *e* and its subcutaneous tissue is carefully dissected off the posterior surface of the auricle and the lateral surface of the mastoid. Care is taken not to expose or injure the auricular cartilage. The skin is then incised *a-b* behind and above the auricle and this incision is carried down to temporalis muscle.

If it is intended to do a tympanoplasty it must be completed before the bone strips are inserted into the cavity. Full-thickness skin *h* (Fig. 10) to be used in the tympanoplasty can be obtained from the posterior surface of the auricle (Fig. 10).

Inserting the Bone Strips

The bone strips soaked in penicillin are now trimmed to size and inserted into the cavity in the manner indicated in Fig. 4(*a*) or 4(*b*) to reconstruct the petromastoid and posterior bony wall of the canal. The latter wall is not reconstructed to its full height; this allows the post-auricular flap to be positioned with ease.

The auricle is released and the postauricular flap *e* (Fig. 11) is carefully positioned to cover all the exposed bone strips. If the superior portion of the flap *e* is found to overlap the grafted area it is carefully split *i* immediately superior to the bone grafts (Fig. 11).

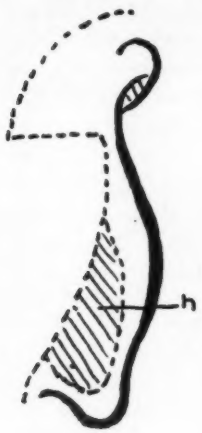


FIG. 10.
h represents the area from which the Woolf graft is taken.

The canal is filled with absorbable gelatin sponge and the post-auricular flap is held in position by packing the canal with a plug of paraffin-gauze.

Closure of the Postauricular Opening

The postauricular opening is closed by carefully suturing, with catgut sutures, the subcutaneous tissues of the auricle, at its free margin, to an area on the surface of the postauricular flap *e* (Fig. 11) which has

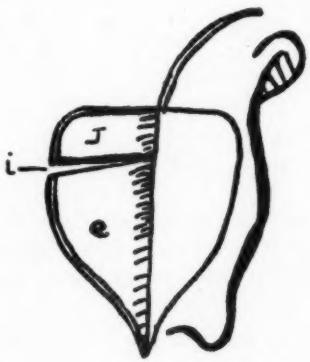


FIG. 11.
Flap *e* split along line *i*. The redundant portion *j* will be resutured to the auricle.

been denuded of its surface layer of dermis along the line corresponding to the original area of attachment of the auricle (Beales and Hynes). Finally, the raw area on the posterior surface of the auricle is covered with a skin graft taken from the thigh.

Secondary Rotation Flap

If the postauricular flap *e* (Figs. 11 and 12) is found to be inadequate in size and mobility to cover the bone strips, it is converted into flap *g* shown in Figs. 12(*a*) and 12(*b*). This flap is easily positioned to provide complete cover to the grafts.

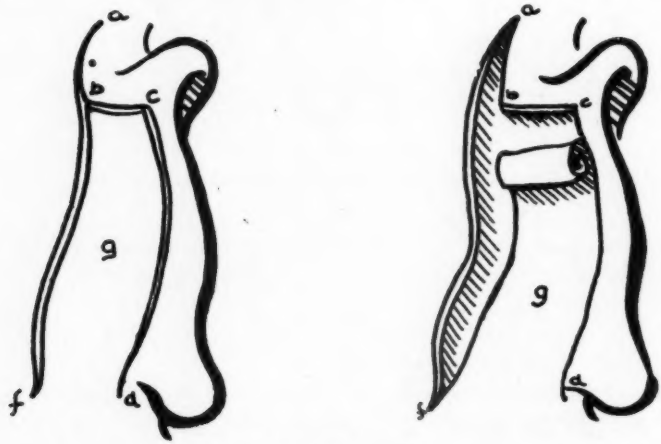


FIG. 12(*a*) and (*b*).

Incision *a-b* is extended through the skin and its subcutaneous tissues along line *a-b-f*. It follows the curvature of the hair line and lies approximately ½ of an inch behind the posterior margin of the re-excavated cavity. By holding flap *e* and continuing the separation of the skin and its subcutaneous tissue from the lateral surface of the mastoid a long broad pedicle flap *g* marked *f-b-c-d* is formed. This flap is nourished by the main trunk of the postauricular artery.

Resuturing of the Flaps

These close the postauricular wound, and the technique for doing this is shown in Fig. 13.

Finally, the raw area on the posterior surface of the auricle and its postauricular region is covered with a full-thickness skin graft taken from the thigh. The graft is held in position by sutures.

2. Using the Postauricular Rotation Flap as a Primary Flap

Planning the Flap

This is illustrated by Figs. 14(*a*) and 14(*b*).

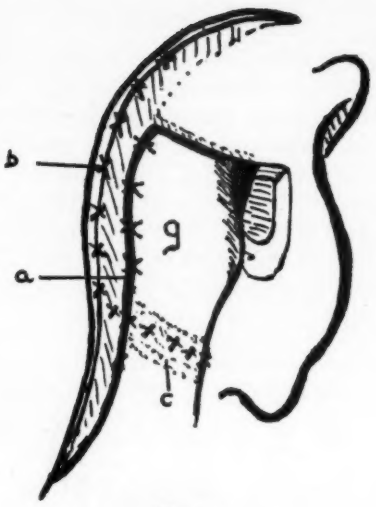


FIG. 13.

Using chromic catgut sutures the posterior margin of flap *g* is sutured to the deep fascia *a* immediately behind the posterior margin of the grafted mastoid cavity. The postauricular opening is closed by suturing, with chromic catgut, the subcutaneous soft tissues of the auricle at its free margin, to the deep fascia or subcutaneous tissue of the skin of the postauricular incision *b*. It is also sutured to a raw area *c* on the pedicle flap, from which the surface dermal layer had been removed.

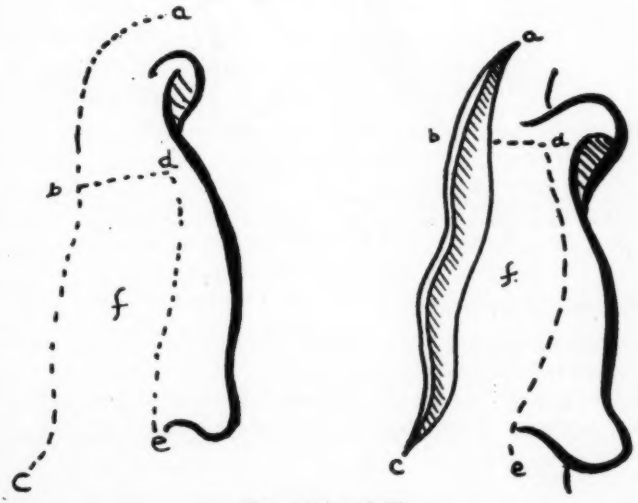


FIG. 14(*a*) and (*b*).

The line of incision *a-b-c-d-e* to construct the flap *f* is outlined with Bonny blue solution. Line *a-b-c* follows the curves of the hair-line and lies ¼ of an inch in front of it. Line *b-d-e* extends on to the posterior surface of the auricle. Point *e* ends at the postauricular sulcus almost at the level of the mastoid tip.

The skin is incised along line *a-b-c*. From *a-b* it extends down to temporalis muscle and from *b-c* down to deep fascia. Do not incise down to bone. The postauricular skin and its subcutaneous tissues are dissected forwards until the posterior border of the old mastoid cavity is reached. Incising the deep fascia down to bone immediately behind the posterior border and extending it along the superior border will expose the old cavity. It will also enable the auricle to be reflected forwards.

Preparing the mastoid cavity, taking the bone strips from the iliac crest, completing the tympanoplasty and inserting the bone grafts is completed in the manner previously described.

FIG. 15.

The rotation flap *f* is constructed by dissecting the skin and its subcutaneous tissue from the underlying structures, within the confines of points *c-b-d-e*. When so doing, care is taken to avoid denuding the auricular cartilage.

Completing the Rotation Flap

Having inserted the bone grafts, the construction of the rotation flap is completed as shown in Fig. 15.

Finally, the flap *f* is positioned and the postauricular wound is sutured as has been described for the secondary rotation flap *g* (Fig. 13).

Conclusion and Summary

The procedure which I have described, utilizing autogenous cancellous bone strips to reconstruct the exenterated petromastoid and posterior bony wall of the external auditory canal, has proved to be an effective

method of obliterating the mastoid cavity and restoring the contour of the external auditory canal. It is applicable to cases requiring modified or radical mastoidectomies, fenestrations and old mastoid cavities, even in the presence of infection.

In the series of 23 cases, the first 7 cases have been observed over a period of 8-12 months. In this series, 22 cases have proved completely successful. One has been a partial failure due to a technical error on my part; I inadvertently failed to provide adequate skin cover for the grafts by cutting the flap too short. Observation has enabled me to assess progress and to establish conclusively (*a*) that the cancellous bone grafts become replaced by new bone which integrates with the mastoid bone even in the presence of infection and (*b*) that the procedure successfully achieves the objectives for which it has been designed.

The advantages of this procedure are many and include the following:

1. It completely obliterates the mastoid cavity.

2. It restores the contour of the mastoid bone and the external auditory canal.

3. It eliminates all the disadvantages arising from mastoid cavity formation.

4. Post-operative treatment is reduced to a minimum, namely: (*a*) packs can be removed painlessly without an anæsthetic; (*b*) the post-auricular and hip wounds are healed by the 7th day, and (*c*) the canal is completely dry and epithelialized by the 3rd-4th week.

The only fair criticism that can be levelled at the procedure is that it necessitates taking bone from an iliac crest. Because of this the patient has to be confined to bed for 14 days. Apart from pain experienced in the hip for 3 days post-operatively, there have been no complications associated with the operation. The results prove that the advantages of the procedure far outweigh the minor disadvantages.

It is suggested that this procedure be used in all cases where a large mastoid cavity would otherwise remain.

Acknowledgements

I should like to record my thanks to the Editors of the *South African Medical Journal* for allowing me to reprint Figs. 1, 2, 3, 4(*a*), 5 and 6; also to Dr. R. L. Kleinman for kindly reading the manuscript of this article. I am indebted to Mr. Martin Singer for taking the iliac grafts, to Drs. N. Smiedt, N. Sher and J. Levine for giving the anæsthetics and to Dr. K. Brauer for taking the radiographs.

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RESULTS OF TYMPANOPLASTIES 1956-1959

By GAVIN LIVINGSTONE and HUGH MILLAR*

SURGERY of the ear, especially in cases of chronic suppurative otitis media, has been changing since 1952, when Zöllner and Wullstein introduced the principle of Tympanoplasty. The term refers to reconstruction of the sound conducting mechanism of the middle ear. It should also imply the total eradication of all disease present. The operations involve the use of full or split thickness skin grafts. The aim of Tympanoplasty is to obtain not only a dry ear, but also an improvement in hearing.

Wullstein has classified Tympanoplasty operations into five types according to the anatomical method used to re-establish sound transmission.

TYPE I.

Plastic repair of tympanic membrane.

TYPE II.

Meatoantrotomy or atticoantrotomy plus tympanic membrane graft over a functioning ossicular chain.

TYPE III.

Columella effect with graft applied either to the head of the stapes or to a polythene strut inserted to replace a damaged stapes.

TYPE IV.

Exteriorization of the stapes footplate with grafting over a Cavum Minorum.

TYPE V.

Plastic operation and subsequent fenestration.

Any standard technique must be modified to meet the existing pathology so that Tympanoplasty operations are rarely identical, and Zöllner and Schuknecht have made further sub-divisions of the five types to allow a greater accuracy of terminology. Tympanoplasty may also be performed for congenital and traumatic defects of the ossicular chain, but

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additional terminologies for these procedures would seem superfluous. For practical purposes the classification into five types appears adequate.

Assessments of Results

The success of a Tympanoplasty operation should be judged both on the improvement in hearing and also on the presence or absence of post-operative discharge.

A. Hearing Improvement.

Several series of Tympanoplasties have been published showing the results to hearing. (Wullstein, Zöllner, Beales, Schuknecht, Proctor, Thorburn). Some of the results have been excellent, but comparison between them is difficult. The criteria for operative intervention differs between each clinic and there are differences of calibration of audiometers.

The American Otosclerosis Study Group recommend that hearing improvement can be best estimated by observing the degree of closure of the air bone gap, and the best way of assessing the improvement in a series of cases is by the percentage closure of this air bone gap. However, the current method of assessing results is to regard as successes cases which obtain a post-operative hearing level of 0–30 decibels loss (i.e., socially adequate hearing). This method excludes the successes in cases with a mixed type of deafness where the post-operative hearing gain may be considerable, but not up to the 30 decibel level. The method also does not take into consideration cases where there is a cholesteatomatous link in the ossicular chain or where a squamous epithelial sac forms a natural tympanoplasty Type IV. Surgery cannot often improve hearing in these cases, but the results are not necessarily failures.

B. Otorrhœa.

Few series include details of the incidence of chronic discharge following Tympanoplasties, although the pre-operative selection of cases will have an important bearing on this result. The pathology in chronic middle-ear disease may be variously described as chronic inactive otitis media, chronic suppurative otitis media, cholesteatoma and chronic mastoiditis. The inclusion in a series of a high percentage of cases with inactive chronic otitis media will necessarily result in more dry ears (post-operatively) than when a high percentage of cases had active suppurative otitis media. The incidence of post-operative discharge can be fairly judged only if the type of pathology in a series is comparable.

Unfortunately pathological classifications of middle-ear disease differ, so clinical observations and operative details should be accurately recorded to ensure that the correct classification is given to each case. There

is a tendency to attempt reconstructive surgery regardless of the type of pathology present. Comparative studies may show that tympanoplasties should not be attempted in certain types of middle-ear disease. For example, the primary aim in cases of unilateral chronic suppurative otitis media is to obtain a dry ear, sometimes these new procedures jeopardize this aim. It is with this danger in view that the following series has been collected.

Tympanoplasty operations have been routine in this department since 1955, in preference to the older type of mastoid operations. The techniques used and the pre-and post-operative management of the cases are basically the same as those described in other series. The operations were performed over a four year period, 1956-1959; they were done mainly by one surgeon (G.H.L.) and the remainder by registrars under his tuition and supervision.

Excepting the TYPE I group, all the cases in this series had active disease or cholesteatoma with chronic discharge which had failed to respond to conservative measures.

Wullstein's operative classification has been used.

Hearing improvement results have been evaluated under three headings:—

1. 0–30 decibel post-operative loss, i.e., socially adequate hearing.
2. Improvement in hearing, but not up to the 30 decibel level.
3. No change.

The average post-operative hearing level for each type has been calculated and also the mean decibel gain. The hearing level has been determined by averaging the hearing loss at the three speech frequencies of 500, 1,000 and 2,000 c.p.s. Audiometry has been carried out in an acoustically treated room with an Amplivox model 61 audiometer calibrated to the American standard.

Each case has been recorded graphically and is represented by a vertical line on the audiogram. A dotted line represents the bone conduction level and, therefore, the ultimate possible hearing improvement. An arrowed line represents the gain or loss in air conduction following surgery, the arrowed end showing the post-operative level, and the horizontal end the pre-operative level. When no gain or loss has occurred air conduction is represented by a horizontal line only. In the TYPE I group, cases in which permanent perforation occurred are marked by a circle.

All cases have been followed up post-operatively for a period of at least six months, and are reviewed at six monthly intervals, and the incidence of discharge noted. Discharge observed on more than one occasion after an interval of three post-operative months has been taken as evidence of failure to heal.

TYPE I—26 cases

Initially simple per-meatal myringoplasties were performed in the Type I group and led to a number of failures. Subsequently a full post-auricular approach with elevation of a posterior meatal skin-flap has been used. (After Wullstein.) This allows a complete inspection of the existing

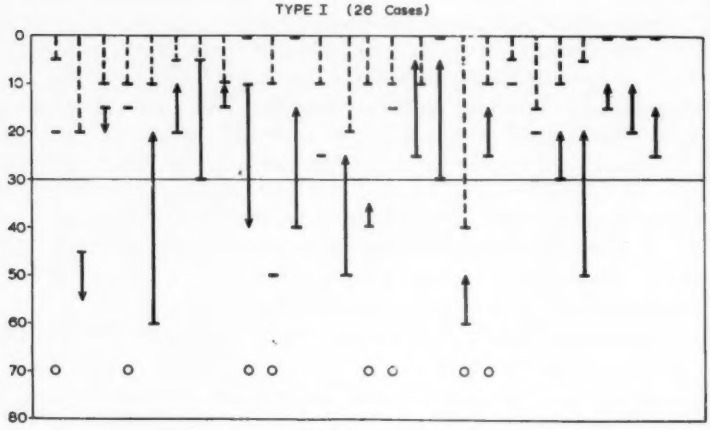

FIG. 1.

FIG. 2.

STATISTICS OF TYPE I CASES

		%
0-30 db	19	73
Improved but not socially adequate	2	8
Not improved or worse	5	19
Average post-operate db level	21 db.	
Average hearing improvement	13·5 db.	
Permanent perforations	8	30

pathology and antral or attic drill holes may be made if indicated. Further, if unexpected pathology is encountered, a Type II or III operation can be immediately embarked upon. It is felt that the use of this approach for all cases may improve the results of Type I cases of which 70 per cent. remained healed with socially adequate hearing. A vein graft applied to the inner surface of the drum is now the method being used.

TYPE II—16 cases

The Type II group comprise the smallest in the series because few cases with active disease have the requisite pathology for this procedure. If there is localized attic cholesteatoma it may be removed and the

tympanum may remain isolated by the sac wall so that there is no need to apply a skin graft. And if there is active disease in the tympanum the long process of the incus is commonly eroded necessitating a Type III procedure. When the ossicular chain is in continuity, grafting cannot be expected to produce a marked hearing improvement.

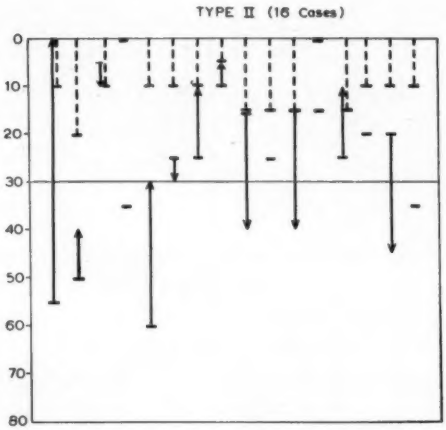

FIG. 3.

FIG. 4.

STATISTICS OF TYPE II CASES

		%
0–30 db.	7	44
Improved but not socially adequate	1	6
Not improved or worse	8	50
Average post-operative db. level	25 db.	
Average hearing improvement	4 db.	
Post-operative otorrhœa	5	31

TYPE III—56 cases

The largest and most successful group was Type III in which nearly 70 per cent. of cases gained an improvement in hearing and the average post-operative hearing loss was only 32 decibels. This is probably due to the ease with which a graft can be placed over the de-epithelialized drum remnant, head of stapes and facial canal. Post-operative otorrhœa was usually due to infection in the mastoid segment. Grafting this area usually is unsuccessful.

The worst results occurred in the Type IV group and, by comparison, are little better than those of radical mastoidectomy. In general, attempts to construct a direct sound pathway to the oval window failed and, in

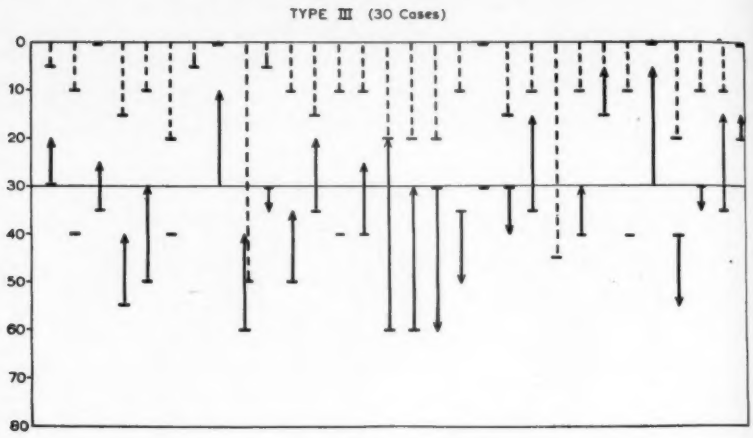

FIG. 5.

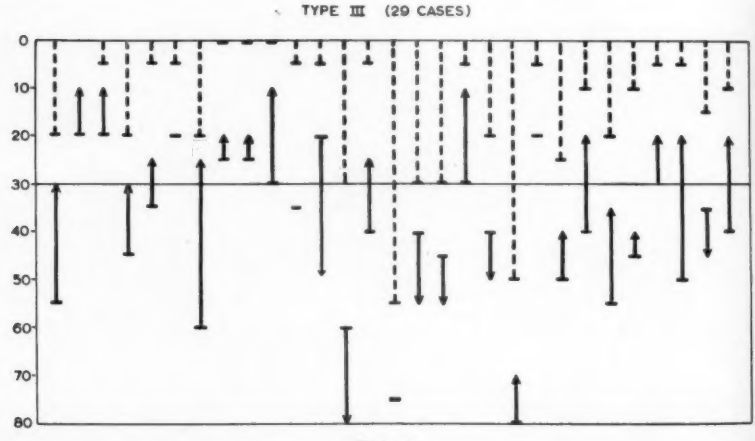

FIG. 6.

FIG. 7.

STATISTICS OF TYPE III CASES

		%
0–30 db.	33	56
Improved but not socially adequate	7	13
Not improved or worse	19	31
Average post-operate db. level	32 db.	
Average hearing improvement	8 db.	
Post-operative otorrhœa	15	26

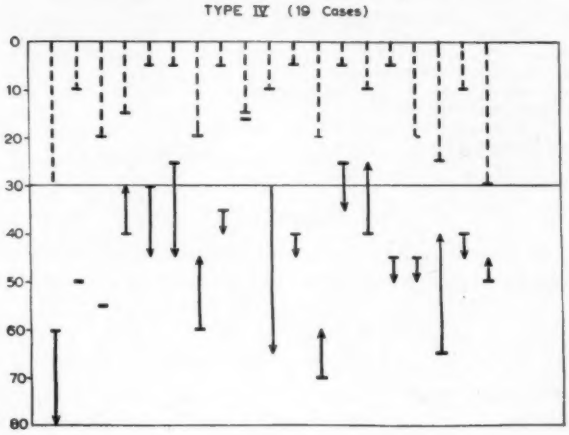

FIG. 8.

this respect, the problem would appear mainly technical. The fact that other authors have gained good hearing improvement in this group supports this supposition. Nevertheless, although the pathology in this group is the most chronic and wide-spread necessitating radical surgery prior to reconstruction, there was no greater incidence of post-operative otorrhœa.

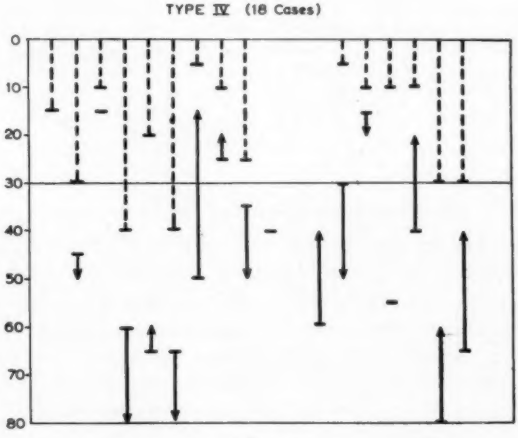

FIG. 9.

FIG. 10.

STATISTICS OF TYPE IV CASES

		%
0–30 db.	7	20
Improved but not socially adequate	9	25
Not improved or worse	21	55
Average post-operative db. level	44 db.	
Average hearing improvement	0 db.	
Post-operative otorrhœa	12	30

Comment

Surgery of chronic middle-ear disease is still a great challenge to otologists. The results presented are regarded as encouraging. Socially adequate hearing has been obtained in a significant percentage of cases in Types I, II and III groups in which the results are comparable to those of other authors (Fig. XI). The results in Type IV group have been disappointing. Adhesions and scarring are the main reasons for failure to produce hearing improvement and if in a dry ear extensive tympanosclerosis is found it is probably wiser to leave the middle ear alone and to fenestrate the lateral semicircular canal.

FIG. 11.

AUTHOR	TYPE I	TYPE II	TYPE III	TYPE IV
Wullstein	96% (322)	86% (196)	80% (490)	68% (280)
Beales	70% (17)	23% (13)	62% (21)	50% (8)
Proctor		63% (57)	52% (44)	28% (60)
Schuknecht	82% (40)	80% (17)	66% (44)	42% (58)
Thorburn	82% (17)	79% (19)	48% (56)	26% (23)
Livingstone	73% (26)	44% (16)	56% (59)	20% (37)

% gaining socially adequate hearing.
Number of cases in brackets.

Comparison of Results of Other Authors

The overall figures show that 70 per cent. of all Tympanoplasties have remained completely dry post-operatively. This suggests that the inclusion of free skin grafts in reconstructing the middle ear mechanism does not increase the incidence of chronic post-operative otorrhœa. However, it is possible that in certain types of pathology, reconstruction should not be attempted as this may jeopardize the chances of a dry ear. In general, the mastoid cavity should be kept as small as possible after all disease has been removed and should not be grafted. Gentle packing appears to produce mastoid cavities which epithelialize completely in the shortest

time. When a large mastoid cavity cannot be avoided there is a place for muscle grafting to reduce the size of the cavity and to provide a healthy bed for epithelialization.

Summary

1. The results of 138 Tympanoplasty operations are reviewed with regard to hearing improvement aud cessation of Otorrhœa.

2. The hearing improvement achieved in Types I, II and III groups was encouraging in that nearly 60 per cent. of these cases acquired socially adequate hearing. Only 20 per cent. of Type IV cases were in this category.

3. In 70 per cent. of all procedures the ear remained completely healed post-operatively indicating that Tympanoplasties are not resulting in any increased incidence of wet cavities.

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EOSINOPHILIC GRANULAR-CELL CYSTADENOMA OF THE LARYNX*

By DONALD HEATH (Birmingham)

ADENOMA of the larynx is a rare disease. In 1920 Moore could trace reports of only 13 cases and when New and Erich (1938) reviewed the 722 benign laryngeal tumours encountered in clinical practice over 30 years at the Mayo Clinic they found only one adenoma. Figi *et al.* (1944) have reported four further cases and Howarth and Chamings (1934) and Ranger and Thackray (1953) have described single examples. In a recent paper Stewart (1960) states that less than 20 laryngeal adenomata have been reported in the past 30 years. The tumours described, almost without exception, were composed of glandular spaces, some of cystic proportions, lined by columnar epithelium that has frequently been mucin-producing and papilliferous. The laryngeal adenoma reported here also showed these histological features initially but recurred as an eosinophilic granular-cell cystadenoma. This occurrence is so rare as to merit its being recorded, for there appear to be only four previous references in the English literature to a cyst of this type in the larynx (Nohteri, 1946; Som and Peimer, 1949; Ellis, 1960; and Pinkerton and Swanson Beck, 1961).

Case Report

A woman, aged 67 years, was admitted to hospital in February 1959 with complaints of hoarseness and inability to control the voice. A polyp was found growing from the right laryngeal ventricle and it was removed at direct laryngoscopy. It consisted of two small nodules of glandular tissue covered by respiratory type epithelium (Fig. 1). The small, irregular, glandular spaces were lined by very tall columnar epithelium some of which was mucin-secreting (Fig. 2). The nuclei were long and oval and tended to be arranged in two rows in a manner reminiscent of an adenolymphoma of salivary gland. While it was clear that the lesion was an epithelial tumour it was difficult to decide whether it was benign or malignant. However, the final diagnosis made was one of cystadenoma of larynx.

Fifteen months later the huskiness of the voice returned and the patient was readmitted to hospital. Clinical examination revealed recurrence of a fleshy, congested tumour again arising from the right laryngeal

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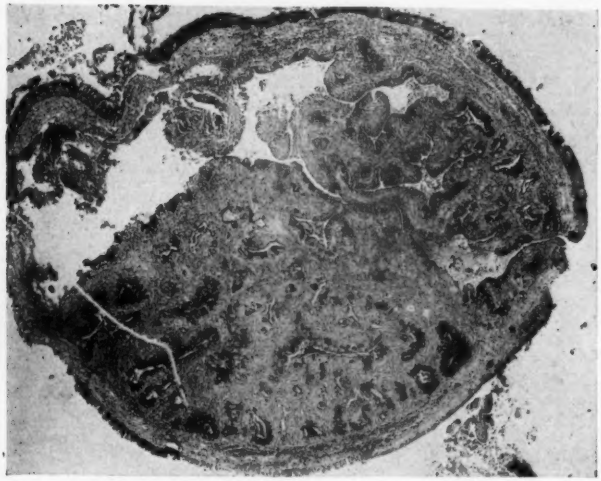

FIG. 1.

Section of the cystadenoma removed in February 1959. It consists of spaces, some of them cystically dilated, lined by columnar epithelium. The tumour is covered by respiratory epithelium. Haematoxylin and eosin. (×23).

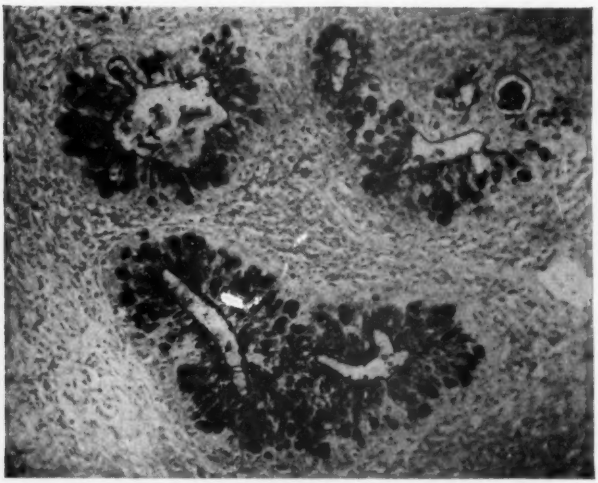

FIG. 2.

Part of the tumour shown in Fig. 1 at higher magnification. The epithelium lining the spaces is mucin-producing and tall columnar in type. Hæmatoxylin and eosin (×135).

ventricle. This was removed at direct laryngoscopy in July 1960. Once again the tumour consisted of cystic glandular spaces covered mainly by columnar ciliated epithelium with small areas of squamous epithelium (Fig. 3). On this occasion, however, the appearance of the glandular epithelium was different for it was columnar and strikingly eosinophilic. The nuclei were small and pyknotic. The cytoplasm was coarsely granular, refractile and brightly eosinophilic (Fig. 4). The cystic nature of the tumour was more pronounced than formerly. Despite the fact that the tumour had recurred there was no histological evidence of malignancy. A diagnosis of eosinophilic granular-cell cystadenoma of the larynx was made. The nature of the component cells is discussed below.

Discussion

The laryngeal adenoma described here is typical of the majority of the recorded examples of this type of tumour both with regard to its macroscopic appearance and its site. It developed in one of the ventricles where mucous glands are numerous. It is known, however, that tumours of this type may grow anywhere in the larynx and extend into the vocal cords. It is also typical of its kind in that it presented as a single pedunculated polyp of about 1 cm. in diameter with a pink, congested colour. Some laryngeal adenomata have been described as grey (Howarth and Chamings 1934). Rarely adenomata in this region are much larger, Figi *et al.* (1944) reporting in one patient two distinct tumours which were respectively 5·5 cm. and 4·0 cm. in diameter. They may also appear as sessile, cauliflower like excrescences but this form of adenoma is much more likely to be multiple than the pedunculated type (Ranger and Thackray, 1953).

The histological appearances of the first polyp removed are also typical (Figs. 1 and 2). Most reported cases have shown the glandular spaces lined by tall, columnar, mucin-producing epithelium as in the present case (Fig. 2), and in some instances the glandular spaces have been cystically dilated to form mucoid cysts so that the tumour has been designated cystadenoma (Figi *et al.*, 1944, Ranger and Thackray, 1953).

The histological appearances of the recurrent cystadenoma were, however, unusual. Its component cells were deeply eosinophilic with a granular cytoplasm and pyknotic nuclei. Cells of this type were originally observed in the tongue, uvula, pharynx and œsophagus of an executed man by Schaffer in 1897 and were termed by him "pyknocytes" on account of the condensed nature of the nucleus. Zimmermann (1927) found them in the salivary glands and trachea. Hamperl (1931, 1950), more impressed with the swollen eosinophilic cytoplasm, called them "oncocytes". He found fully formed and transitional forms in the pancreas and other organs. Both he and Nohteri (1946) found that

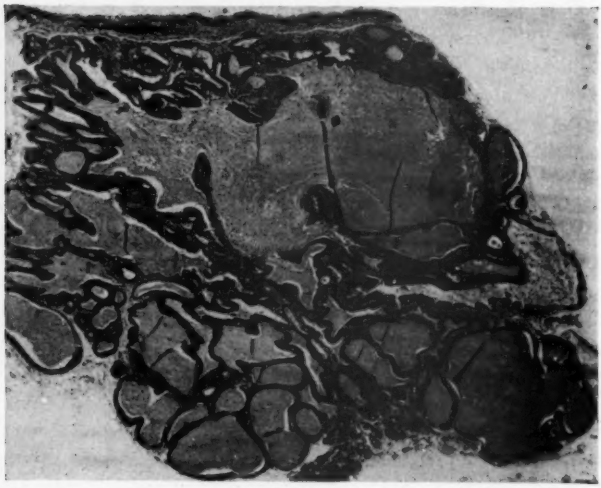

FIG. 3.

Section of the recurrent eosinophilic granular-cell cystadenoma. The tumour still consists of glandular spaces, some of which are cystically dilated. The tumour is covered by respiratory type epithelium. Hæmatoxylin and eosin (×23).

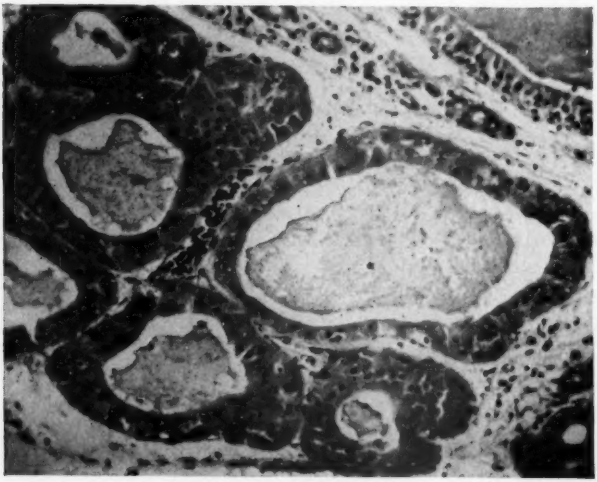

FIG. 4.

Part of the tumour shown in Fig. 3 at higher magnification. The epithelium lining the spaces is strikingly eosinophilic and the nuclei are small and pyknotic. Hæmatoxylin and eosin (×150).

oncocytes were absent before the age of 50 years and were always found in patients over 70 years old. They regarded the swollen eosinophilic cells as an expression of degenerative age change. Pinkerton and Swanson Beck (1961) have retained the term oncocyte and called these cystadenomas "oncocytic cysts". In view of the fact that oncocytes are regarded as degenerate cells of middle aged and elderly people, it is of considerable interest that all eight oncocytic cysts reported (including the present one), have been in patients over 50 years of age. Willis (1953) believes that there are no grounds for regarding these eosinophilic cells as a special kind of epithelium and suggests that the term "oncocytoma" and presumably "oncocytic cyst", should be discarded.

The recurrence of the present tumour is itself unusual. Figi *et al.* (1944) found no evidence of recurrence, for instance, in their four laryngeal cystadenomata, even after 8 years in one case. The oxyphilic adenoma reported by Ellis (1960) had not recurred after an interval of 6 years. Most patients with a laryngeal adenoma, including all four with oncocytic cysts described by Pinkerton and Swanson Beck (1961), complain of hoarseness as described above, but if the polyp has an unusually long pedicle it may stimulate coughing (Figi *et al.*, 1944). Infected laryngeal polyps including adenomata are œdematous and swollen and may give rise to dyspnœa (Figi *et al.*, 1944). Most pedunculated tumours of the larynx may be removed easily at laryngoscopy as in this case but the rare diffuse variety may require laryngofissure (Ranger and Thackray, 1953).

Summary

A woman of 67 years complained of hoarseness. A benign polyp was found and removed at laryngoscopy. It proved to be a cystadenoma composed of glandular spaces lined by tall columnar, mucin-producing epithelium. Fifteen months later it recurred and on this occasion had the histological appearances of an eosinophilic granular-cell cystadenoma.

Acknowledgement

I wish to acknowledge the permission of Mr. A. J. Moffett, F.R.C.S., Consultant Surgeon, The General Hospital, Birmingham, and to Dr. D. B. Brewer, M.D., to publish this case report.

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CLINICAL RECORDS

FATAL CONGENITAL STENOSIS OF THE TRACHEA

By RUSSELL DALLACHY (Glasgow)*

TRACHEAL obstruction at birth and during the neonatal period is rare compared with laryngeal obstruction. When it does occur, it is due most often to some extrinsic cause, which produces deformity of a normal tracheal wall. In the patient described in the present paper, there was marked narrowing of the trachea immediately below the larynx, due to thickening of the cricoid cartilage. The clinical features and special investigations are described, possible treatment discussed, and the few similar cases found in the literature are reviewed.

Case Report

The case reported was the third pregnancy of Mrs. A. M., a former midwife. She was married at the age of 29 in 1950. The first pregnancy ended as a twin abortion in the 4th month, in 1953. The second terminated at the end of the 1st month, in 1957. The L. M. P. of the third pregnancy was on *7.9.59*, and the patient was admitted to hospital at the 8th week for prolonged rest because of her previous history of abortions and because her age was then 39; there was no sign of toxæmia. She received no hormone treatment, and took her discharge after 3 weeks in hospital. Her blood group was O, Rh (D) positive. Apart from appendicectomy at the age of 9 years, there had been no major illness.

Labour started on *24.5.60* (10 days later than expected), and a mature, live, male child, weighing 6 lb. 9 oz. (2,977 g.) was born by vertex presentation. Labour lasted 4 hours 15 minutes, and trichlorethylene with air was given intermittently. A succenturiate placenta with a velamentous insertion of the umbilical cord was complete, apparently healthy, and weighed 1 lb. 9 oz. (709 g.).

The child (blood group O, Rh (D) positive) was limp at birth and cyanosed. Respiration, although starting within a minute of birth, was accompanied by marked indrawing of the subcostal and sternal areas. Crying occurred 7 minutes after birth and was frail and whimpering. Suction by a soft rubber catheter was applied to the upper respiratory passages, then a laryngoscope was passed and a small amount of mucus was aspirated; finally about 4 ml. of mucus was extracted through a gastric tube. Oxygen was given, by a face mask at first, and thereafter in an incubator; Vitamin K, 1 mg., was injected intramuscularly. Cyanosis diminished appreciably, but the indrawing of the chest wall on inspiration continued unchanged.

A pædiatrician first saw the infant when it was just over 8 hours old. The mouth was normal. There were vigorous respiratory movements, with indrawing of the chest wall; air movement through the nostrils seemed diminished, but a

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rubber catheter could be passed 6 inches (150 mm.) into them. The percussion note was resonant over all lung zones, but air entry was diminished; there were no adventitious sounds. Radial and femoral pulses were normal. The apex beat was neither visible nor palpable; a diffuse, loud, præcordial systolic murmur was heard. The anterior edge of the liver was below the right costal margin: the spleen was not felt. The anterior fontanelle was tense. Muscle tone was normal.

FIG. 1.

Skiagram of chest on 1st day of life. Heart is globular, with right border almost reaching the right mid-clavicular line. C-T ratio, 0·55:1. No intra-thoracic cyst or tumour.

Radiographic examination. The cardiac shadow was central and globular; although the right border extended almost to the right mid-clavicular line, this is not outside the range of normality for neonates. The cardio-thoracic ratio was 0·55: 1 (normal range for infants up to 3 weeks old is 0·60: 1 to 0·50: 1). Both lungs were aerated, there was no segmental lesion, and no sign of intra-thoracic cystic or solid neoplasm (Fig. 1). On *27.5.60*, iodized oil, introduced into the œsophagus by catheter, flowed freely into the stomach with neither holdup nor indentation on screening; films showed the whole of the œsophagus outlined and confirmed that there was no obstruction (Fig. 2).

Treatment and progress. Streptomycin, 50 mg. intramuscularly, was given 4 times daily. On the day after birth, cyanosis was noted only when the child cried, although the vigorous but ineffectual respiratory movements persisted. On the following day, slight stridor was noted, and the respiratory murmur became slightly louder when the mouth was held open and the tongue held forward; there was no improvement when the infant was put in the prone

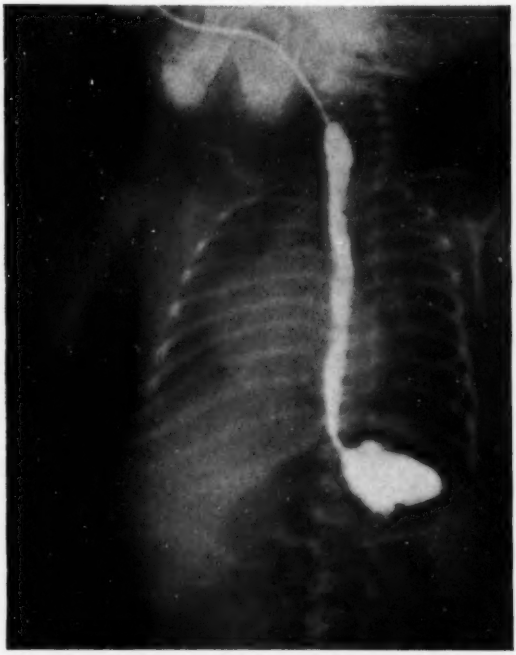

FIG. 2.
Œsophagus outlined by iodized oil, which is flowing freely into the stomach.

position. Lumbar puncture and right and left subdural taps produced cerebrospinal fluid, which had no increase in cells, contained no acid fast bacilli, gave no growth on culture, and showed no biochemical abnormality. On *27.5.60*, subcostal and sternal retraction on inspiration was unchanged and cyanosis occurred when the child was handled during clinical examination. The liver edge was now 45 mm. below the costal margin and the tip of the spleen was palpable. Direct laryngoscopy (Dr. H. M. Urquhart) showed no obstruction; the larynx and the vocal cords moved normally: there was bulging of the left wall of the trachea just below the larynx, with deviation of the trachea to the right; the lumen of the trachea appeared patent.

On *28.5.60*, the patient was seen by a thoracic surgeon (Mr. F. J. S. Gowar). The diagnosis of tracheal obstruction was accepted. It was considered that the obstruction was probably too low for tracheotomy to be performed with any hope of success. On *29.5.60*, cyanosis on handling was much more readily provoked, and the systolic murmur was much louder and was heard over the whole of the left side of the chest. Direct laryngoscopy was again carried out the next day, and an attempt to pass a fine nylon tube into the trachea failed; the tube was held up just below the level of the larynx. On *31.5.60*, the child was cyanosed at rest and after the mid-day feed suddenly collapsed and died.

FIG. 3(*a*).

(A) Tongue, pharynx, larynx and trachea, removed at necropsy; opened in sagittal plane of posterior wall of pharynx, larynx and trachea to show localized narrowing of trachea, due to thickening of anterior and lateral portions of the cricoid cartilage (× 1½).

Necropsy

Necropsy was performed 18 hours after death.

External appearances. Mature male infant, weighing 2,862 g., with a crown-heel length of 475 mm. Moderate diminution of the elasticity of the skin was the only abnormal feature.

Respiratory System. The pleural cavities were healthy. The intra-thoracic lymph nodes were not enlarged. The mouth, tongue and pharynx showed no abnormality. The larynx was normal and was not compressed. The trachea showed a smooth constriction, due to thickening of its wall immediately below

the larynx; the thickening was due to hypertrophy of the cricoid cartilage opposite the isthmus of the thyroid (Figs. 3 and 4); the overlying mucous membrane was intact; a small amount of yellowish mucus was present distal to the constriction, which was less than 1 mm. in diameter, and allowed a fine probe to pass with difficulty; the remainder of the trachea and the bronchi were healthy, with fully formed cartilages, which maintained the patency of the upper respiratory passages. The lungs were aerated and appeared normal There was neither compression nor distortion of the respiratory tract from extrinsic causes.

FIG. 3(*b*).

(B) Outline diagram of (A). T, tongue; E, epiglottis; A, arytenoid cartilages; C, lamina of cricoid cartilage (cut surfaces). Site of narrowing is shaded in solid black.

Circulatory System. The pericardial sac was intact and healthy. The heart (31 g.) showed no external abnormality, and no abnormality of the great vessels was noted; there was a large atrial septal defect, 10 × 8 mm., without fenestration; no other congenital abnormalities were present.

The *abdominal cavity* showed no abnormality; the liver showed neither gross nor microscopic pathological change.

The *skull* and *brain* showed nothing of note.

Microscopic Examination

Larynx and *trachea.* Sagittal and para-sagittal sections through the upper part of the respiratory tract reveal that the obstruction at the upper end of the trachea is a greatly thickened cricoid cartilage. The thickening is mainly

in the central anterior part, so that the narrowed entrance to the trachea is close to the posterior wall. The cartilage is of the normal hyaline type, with many cells uniformly distributed, and not in groups of 2 or 4; this is the normal finding in infancy. The thickened cartilage is covered by normal respiratory epithelium, and there is no other structural abnormality (Figs. 5 and 6).

Lungs are aerated and a few alveoli appear to be moderately overdistended.

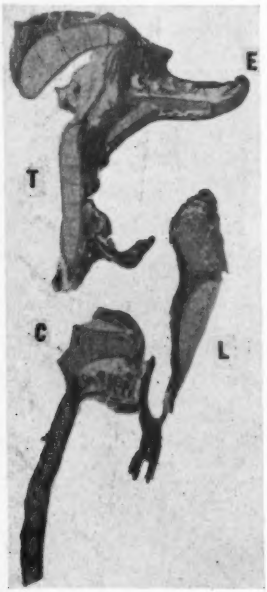

FIG. 4.

Sagittal section of base of tongue, epiglottis, larynx and upper portion of trachea, showing hyperplastic anterior portion of cricoid cartilage projecting posteriorly and obstructing the trachea at its commencement. Part of the lamina of the cricoid cartilage (cut obliquely) is seen on the right. T, thyroid cartilage; C, hypertrophied anterior portion of cricoid cartilage; E, epiglottis; L, Lamina (posterior portion) of cricoid cartilage. (H and E × 3).

Discussion

The main clinical problem in the present case was to determine the cause of the gross obstruction to respiration, and this could not be done during life. There was no abnormality in the nose, pharynx or larynx. On direct laryngoscopy, the left wall of the trachea, immediately below the larynx, seemed to be bulging medially, as though pushed by a mass in the mediastinum. A cyst in the mediastinum is usually the result of abnormal budding of the entodermal tube, and it may be lined by bronchial, gastric or intestinal epithelium (Potter, 1952). Signs may be evident in infancy or may be delayed for many years. If this had been the cause of the trouble in the present case, then it was likely that compression of the trachea would extend to the carina and might possibly

involve the bronchi, so that even a low tracheotomy would fail to bypass the obstruction. Neither a cystic nor a solid mediastinal mass was demonstrable radiographically.

The widespread præcordial systolic murmur was considered to be due to an atrial septal defect, and this abnormality was in fact present. It was thought that one or more associated congenital abnormalities of the heart or great vessels

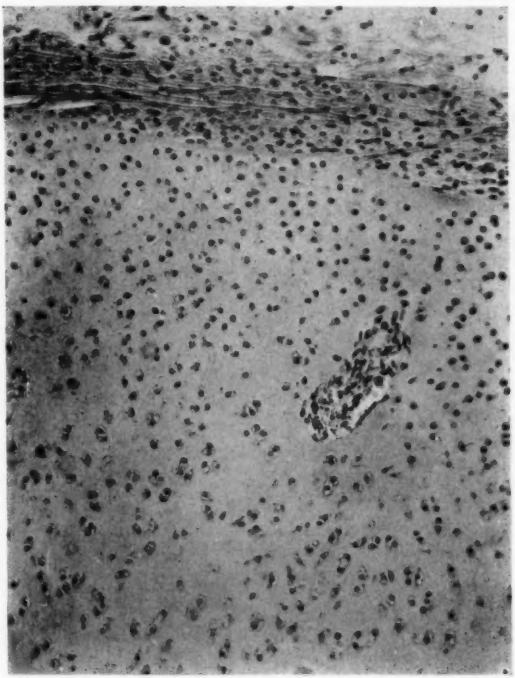

FIG. 5.

Section of the hyperplastic portion of cricoid cartilage with the fibrous perichondrium at the top and active normal cartilage cells evenly distributed deep to the perichondrium (H and E × 120).

might be present. A congenital malformation of the great vessels which causes compression of the lower part of the trachea is the persistence of the right fourth aortic arch with the formation of a right aortic root. The transverse portion of the arch of the aorta divides into right and left roots which encircle the tracheal bifurcation and the œsophagus, and join together posteriorly to form the descending aorta. Compression of these two structures by this vascular ring may cause stridor, chronic cough, dysphagia and feeding difficulties (Wolman, 1939: Morison, 1952: Potter, 1952). No deformity of the œsophagus was demonstrable radiographically in the present case, and the child fed well, with no difficulty in swallowing. Furthermore, the symptoms are generally

mild immediately after birth and become progressively more severe, whereas the patient described had marked difficulty with respiration from the start.

Two intrinsic causes of tracheal obstruction are (i) subglottic webs or redundant folds of mucous membrane, and (ii) defective formation of tracheal cartilages. In the patient described by Glaser *et al.* (1935), redundant folds of tracheal mucous membrane gave rise to dyspnœa, which was first noted at 7 weeks. Subglottic webs are a well known cause of stridor, but this is usually delayed until childhood or even adult life, and only one example was found

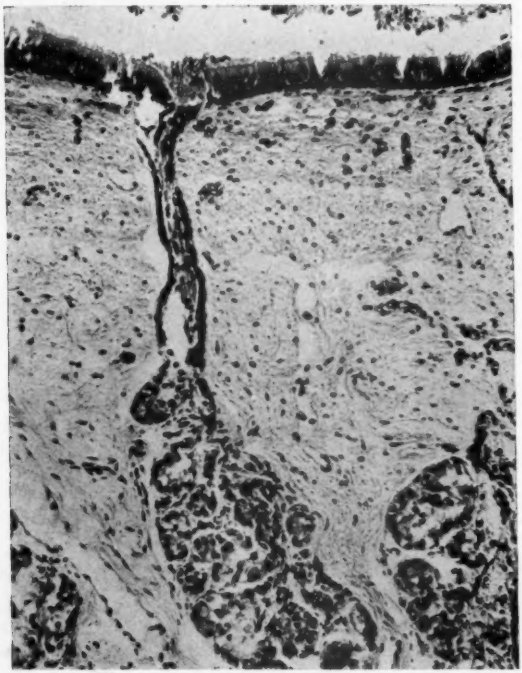

FIG. 6.

Section of the normal, columnar mucin-secreting epithelium (top,) overlying the hyperplastic cricoid cartilage (not shown); numerous mucous glands are present in the sub-mucous layer (H and E × 120).

among 30 cases of congenital stridor reported from the Mayo Clinic by Kennedy and New (1931); at the Ear, Nose and Throat Department of the Victoria Infirmary, Glasgow, subglottic web has been found in members of three generations of one family (Howie *et al.*, 1960). When there is defective formation of the tracheal cartilages, the tracheal walls collapse inwards since there is nothing to maintain the patency of the tube, which is tightly closed during inspiration (Wolman, 1941). In the present case, this was considered the most likely cause of the signs.

The actual cause of the obstruction was constriction of the upper end of the trachea immediately below the larynx by a thick cartilaginous ring. The nature of the obstruction was hyperplasia of the cells of the cricoid cartilage. The portions of the cartilage involved by the proliferative process were the anterior and lateral walls, while the posterior portion (the lamina) was unaffected. The external surface of the cricoid with its enveloping perichondrium was normal and increase in the thickness of the wall was directed inwardly, so that the lumen was diminished (Fig. 7). If the lesion had been a true tumour (i.e. a chondroma),

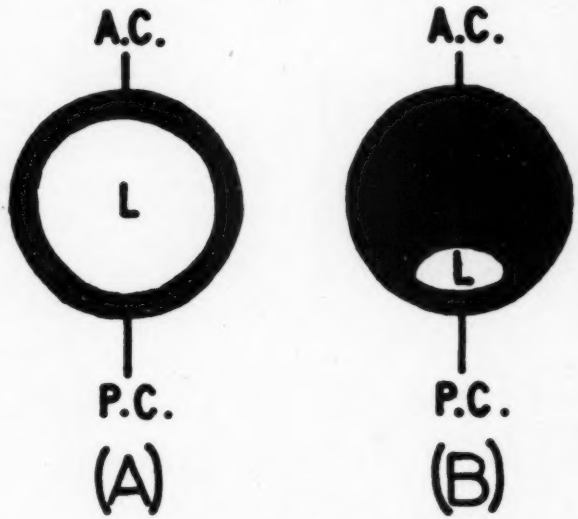

FIG. 7.
(A) Cross section of normal cricoid cartilage (diagrammatic).
(B) Cross section of cricoid cartilage in case reported. A.C., anterior part of cricoid; L, lumen of trachea; P.C., posterior part (lamina) of cricoid.

there would have been a fibrous septum or capsule close to the line where the inner wall of a normal cricoid cartilage would be expected and enclosing the growth. Since there was no such capsule present. it seems best not to give the condition a name, but to describe it as a thickening due to hyperplasia of the cartilage cells of the anterior and lateral walls, with the increased bulk projecting into the lumen of the trachea. There is no apparent reason why the lamina (posterior portion) of the cricoid cartilage should have been excluded from this disordered process of growth.

In Wolman's patient, the trachea was funnel-shaped, with narrowing most marked at the bifurcation; the membranous portion of the trachea was absent; the main bronchi showed stenosis; tracheotomy failed to relieve the stridor, and death occurred when the child was aged 10 weeks. Wolman found 10 cases of congenital tracheal stenosis in children, all recorded in either the French or German literature between 1842 and 1938. One was stillborn, one had attacks

of nocturnal dyspnœa, which eventually ceased at the age of 7 years, following tracheal bouginage and tracheotomy, and the remainder died between the ages of 4 days and 17 months. All but 3 of these 11 cases had some associated congenital abnormality, but none had an interatrial septal defect, as in the present case.

The actual deformity in these 11 cases was different from that reported here. In 3 patients, there was a constriction limited to a short segment of the

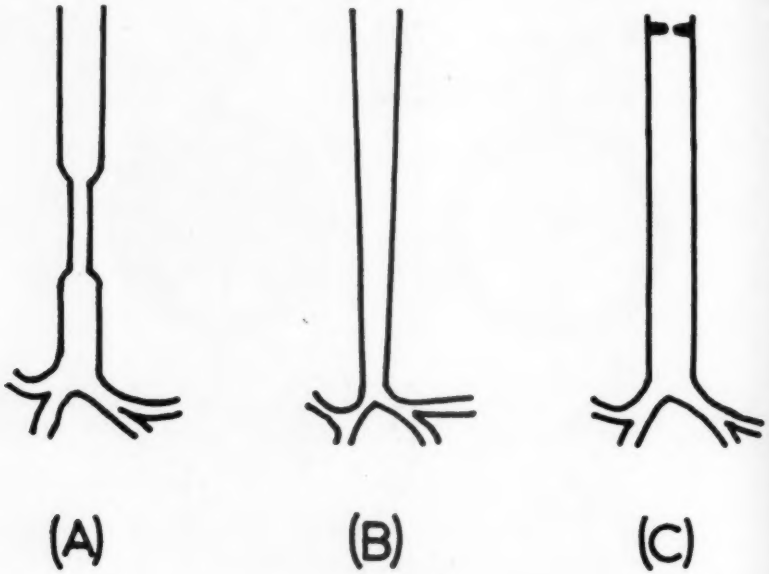

FIG. 8.

Three types of stenosis of trachea (modified from Fig. 4 of Wolman, 1941).

(A) Localized or segmental stenosis.

(B) Generalized, progressive stenosis (funnel trachea), with narrowing of the main bronchi.

(C) Localized stenosis in the present case, due to hyperplasia of anterior and lateral parts of the cricoid cartilage.

trachea, the remainder (including the part immediately below the larynx) being of normal diameter. The other 8 patients showed progressive narrowing of the trachea from above downwards, often with deformed or absent cartilaginous rings and absence of the membranous portion. In the patient described here, the only abnormality was a marked eccentric increase in the size of the cricoid cartilage, the enlargement being directed posteriorly, so that there was only a tiny lumen at the upper end of the trachea close to the posterior wall; these findings are shown diagrammatically in Figs. 7 and 8.

The only similar cases to that reported here are noted by Potter (1952) in her textbook dealing with fœtal and neonatal pathology. She mentions three

infants in whom a solid bar of cartilage, about 3 mm. thick, extended across the larynx immediately below the vocal cords, with a tiny hair-like opening connecting the respiratory tract above and below the stricture. An example is shown in her Fig. 273a. The mucosal surfaces were covered by normal intact tracheal epithelium above and below the bar, and the lungs were normally developed.

If the correct diagnosis could have been made during life in the present case, and it had been realized that the lesion was localized to the upper extremity of the trachea, tracheotomy would have relieved the obstruction temporarily, though it is doubtful if a permanent cure of the condition would have been possible. Tracheotomy was considered, but it was thought probable that the obstruction involved the trachea along too much of its length for any reasonable hope of success.

Summary

Gross respiratory obstruction in a mature newborn infant caused death on the eighth day. At necropsy, there was gross thickening of the cricoid cartilage.

A large atrial septal defect, suspected during life, was also present. The mother (aged 39) had neither toxæmia nor infections during the pregnancy.

Difficulties in diagnosis and problems of treatment are discussed.

The few similar cases recorded are reviewed briefly.

Acknowledgements

I am indebted to Dr. Patrick Macarthur, Pædiatrician to the Northern Regional Hospital Board, for access to the clinical records and for permission to publish; to Mr. J. Hunt for Figs. 1-3; and to Mr. H. C. Gray for Figs. 4-8. I am grateful to Dr. W. B. Davis and Mr. I. C. Simpson for much helpful advice and criticism.

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A CASE OF OSTEOMYELITIS OF MAXILLA TREATED WITH CELBENIN

By H. M. PARSONS (London)

THE infant C.J. was admitted to the E.N.T. Unit at Hither Green Hospital as a typical case of Osteomyelitis of the Left Maxilla.

The mother having had a staphylococcal breast abscess, the infant of six weeks old developed a sore mouth two days prior to admission. With constant crying and poor suckling a swollen left eye was said to have appeared twenty-four hours prior to admission.

Oedema + Exophthalmos

Purulent discharge

FIG. 1.
Monday

On examination the infant was flushed, mildly dehydrated and pyrexial to 102 deg. F. The left eye was completely closed with inflammatory œdema, the palate on the left side was œdematous and the left alveolus was discharging pus between the lateral upper incisor and canine teeth.

Large doses of crystalline penicillin were prescribed, but after twelve hours the condition was still deteriorating.

Coma had developed, the neck was completely stiff and the left eye was protruding much more. The skin by the inner canthus showed early signs of sinus formation.

Discharging sinuses

FIG. 2.
Monday

Cavernous sinus thrombosis was diagnosed.

Celbenin—100 mgms—intramuscularly at six-hourly intervals was prescribed and after only three injections the condition rapidly improved. The infant was virtually normal within 48 hours. The drug was continued for four weeks.

No sequelae have since developed, except that the lateral upper left incisor tooth has been extruded.

FIG. 3.
Thursday

Celbenin is Sodium 6. (2·6 dimethoxy benzamido) penicillinate monohydrate. It is a soluble crystalline powder suitable for intramuscular injection. The substance is active *in vivo* against penicillinase producing staphylococci.

In recording this case I should like to express my gratitude to the Nursing Staff and my colleagues at the E.N.T. Unit at Hither Green Hospital.

2 Upper Wimpole Street,
London, W.1

BILATERAL TUMOURS OF THE PAROTID GLAND

By H. DIAMANT, C. M. ENEROTH and T. GEJROT (Stockholm)*

VERY seldom encountered are bilateral parotid tumours, other than papillary cystadenolymphomata, approximately 5 to 10 per cent. of which are of bilateral occurrence (Foote and Frazell, 1953). Kaliampetsos and Bonmann (1959) on surveying earlier literature found that aside from papillary cystadenolymphomata, bilateral parotid neoplasms were no more common than was to be expected statistically. Papillary cystadenolymphomata are considered to be of multiple origin (Shaw and Friedmann, 1959), and their substantially higher incidence of recurrence—12·2 per cent., as compared with 4·8 per cent. for other benign tumours (Frazell, 1954) seems to support this belief.

Although mixed fibromyxo-epithelial tumours constitute the largest group of neoplasms occurring in the parotid region, bilateral mixed tumours are exceedingly rare (Baker, *et coll.*, 1956); indeed, Foote and Frazell (1953) found not a single case among their series of 760 patients—a fact which in their view militates against the theory of multiplicity. In a ten-year series comprising about 700 parotid neoplasms observed at Karolinska Sjukhuset, only one case with bilateral mixed tumours was encountered (Diamant *et coll.*, 1961).

In the opinion of Foote and Frazell (1954) the multiple tumours found at repeat operations are attributable to insufficiently extensive surgery at the first operation rather than to primary multiplicity.

One tumour that appears to be quite specific for the parotid gland is acinic cell carcinoma, described by Masson as early as 1924. It is considered to be moderately malignant and comparatively rare, but its seeming rarity is partly due to the fact that tumours of this type are often recorded simply as parotid adenomata without exact classification (Foote and Frazell, 1954). However, they are commoner than is generally thought, as indicated, e.g., by the fact that acinic cell carcinomata comprise 2·5 and 3·2 per cent. respectively of the parotid neoplasms in the two largest series reported thus far (Foote and Frazell, 1953; Beahrs *et coll.*, 1960). Since neither of those series includes bilateral tumours of acinic cell carcinoma type, we feel that a report of two personal cases might be of interest.

Personal Cases

Approximately 700 parotid tumours were surgically treated at the otolaryngology department of Karolinska Sjukhuset during the period from 1950 to 1960. They included three bilateral cases—one with benign mixed tumours and the other two with acinic cell carcinomata.

One of the two latter cases was a 71-year-old man who had, anterior to his right ear, a slow-growing tumour of ten years standing. An almond-sized neoplasm

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of the parotid region had been excised at his local hospital. Since the pathologist's report showed acinic cell carcinoma, the patient was referred to Karolinska Sjukhuset, where examination disclosed an almond-sized fluctuating abscess at the site of the surgical scar. The patient said that one year previously he had also felt a hard, painless, pea-sized mass below the left ear. Following preoperative radiotherapy, right parotidectomy was done. In the surgical specimen was found tumour tissue of the same type as that at the original operation. Shortly

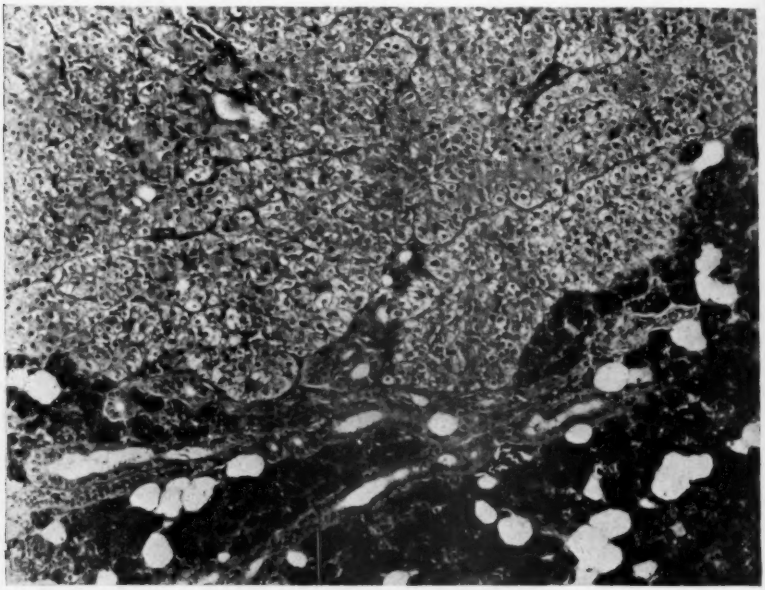

FIG. 1.
Acinic cell carcinoma (150 x)

afterwards left parotidectomy was undertaken as well. Histopathologic examination revealed acinic cell carcinoma of a type similar to that on the right side (Fig. 1). Following consultation with a radiologist, postoperative radiotherapy was decided upon.

The second patient was a 55-year old woman who, one year before admission, had noted a resistant mass below each ear. At her local hospital a biopsy was taken on the right side and showed multiple lesions of salivary gland adenoma. Following preoperative bilateral teleradium treatment, the palpable tumours were excised. On conclusion of the operation a radium tube was inserted in the left side. Histopathologic examination showed multiple bilateral adenomata of the salivary glands. About six months after discharge, a small tumour recurred on the left side and was excised. On re-examination of the histological specimens it was found that all of them justified the diagnosis of acinic cell carcinoma. (Fig. 2.)

Conclusions

In a personal series comprising 700 cases of parotid tumours, two cases of bilateral acinic cell carcinoma were encountered. Although this type of tumour constitutes approximately 3 per cent. of all parotid neoplasms, no bilateral case has previously been reported. The diagnosis of acinic cell carcinoma, it should be pointed out, may be overlooked at microscopic examination and the tumour classed simply as parotid adenoma without specification of the type. This is

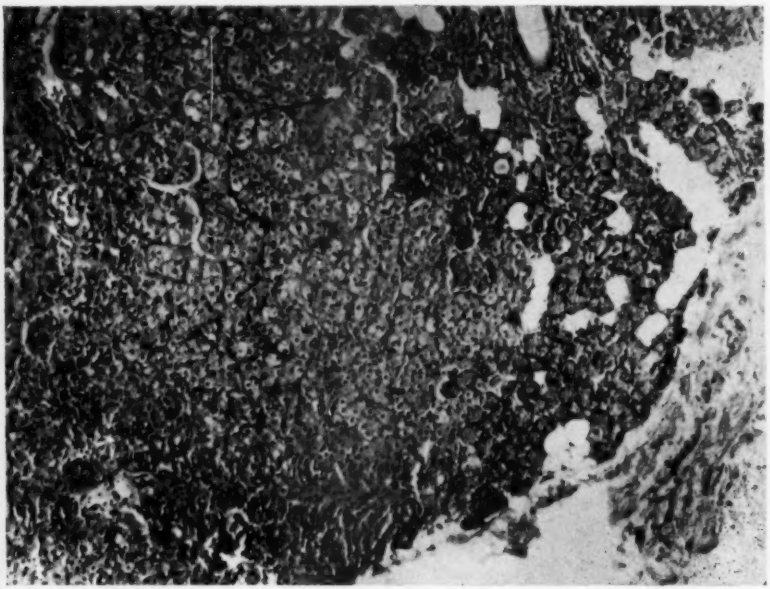

FIG. 2.
Acinic cell carcinoma (150 x)

illustrated by one of our cases, in which the excised tumours were first regarded as multiple parotid adenomata. It was not until later re-examination that acinic cell carcinoma was diagnosed.

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CLINICAL NOTE

A PRELIMINARY REPORT ON FUNGAL INFECTIONS OF MASTOID AND FENESTRATION CAVITIES

By G. D. L. SMYTH* (Belfast)

TOWARDS the end of 1956 it was noted that in a proportion of patients receiving prolonged treatment for infected mastoid and fenestration cavities, fungi were repeatedly isolated. Since most of the cases had no visible evidence of fungal infection (such as sporing heads or other fungal structures) it was decided to investigate the incidence, identity and aetiological rôle of fungi in infected aural cavities. At the same time 80 patients were selected for trial with three anti-fungal drugs.

A total of 3,000 specimens from 350 patients have so far been examined. The fungi observed and the numbers of patients from whom they were isolated were as follows:

Aspergillus niger	27
Aspergillus flavus-oryzae	11
Aspergillus terreus	25
Aspergillus glaucus	4
Aspergillus fumigatus	9
Candida albicans	20
Penicillium sp	2
Scopulariopsis	1

From the work completed to date, several conclusions can be arrived at. It is clear that these fungi are actively associated with some infections of mastoid and fenestration cavities. *Aspergillus niger* and *A. fumigatus* infections appear to be regularly accompanied by pain. *A. niger* and *A. flavus-oryzae* infections are difficult to eradicate permanently. Some laboratory evidence has been obtained which indicates that a large majority of fungal infections commenced during the topical use of broad spectrum antibiotics. The probability of fungal implication in an infection appears to be proportional to the duration of the antibiotic treatment.

A full report of this work will be published at a later date.

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Belfast

*From E.N.T. Departments of the Royal Victoria, Belfast City and Belfast Ophthalmic Hospitals and the Department of Microbiology, The Queen's University of Belfast.

GENERAL NOTES

TEMPLE UNIVERSITY SCHOOL OF MEDICINE

PHILADELPHIA 40. PA.

A "Short course in Audiology" is offered several times each year by the Section of Audiology, Department of Otorhinology, Temple University School of Medicine, Philadelphia 40, Pa.

The course will be one week in length and will encompass theoretical and clinical aspects of audition. Hearing testing and interpretation will be stressed. Office procedures, equipment and Industrial Audiometry will be discussed.

Further information may be obtained by writing to Philip E. Rosenberg, Ph.D., Director, Section of Audiology, Temple University Medical Center, 3401 North Broad St., Philadelphia, Pa.

SIXTH INTERNATIONAL CONGRESS OF AUDIOLOGY

THE sixth International Congress of Audiology will be held in Leyden, The Netherlands, from September 5th to 8th, 1962.

President: Prof. Dr. H. A. E. van Dishoeck;

Secretary Drs. A. Spoor.

In the programme are planned three round-table talks, with associated and free papers.

The subject of the round tables are:

"Frequency analysis of the normal and pathological ear."
Moderator: Prof. Dr. G. von Békésy.
"Central deafness in children."
Moderator: Prof. Dr. J. M. Tato.
"Psychogenic deafness and simulation."
Moderator: Prof. Dr. H. A. E. van Dishoeck.

Official languages of the Congress are: English, French, German and Spanish.
Working languages will be: English and French.

The address of the secretariat is:

Ear-Nose-Throat Department,
Academisch Ziekenhuis,
LEIDEN (The Netherlands).

General Notes

SEMON LECTURE, 1961

The University of London announce that the Semon Lecture will be delivered by Mr. C. P. Wilson at the Royal Society of Medicine on Thursday, November 2nd, 1961, at 5 p.m. The title of the lecture will be "Pharyngeal Diverticula, their Cause and Treatment".

Mr. John F. Simpson will be in the Chair.

BRITISH OTOLARYNGOLOGICAL CONGRESS

It is proposed to hold the first British Otolaryngological Congress in Great Britain in the summer of 1963 and an organizing committee is being set up for the purpose. It is expected that in addition to sessions for formal papers there will be a number of seminars on various topics and revision courses conducted by known authorities.

The Congress will probably last for five or six days.

Further details concerning time and place will be notified in this Journal in due course.

BACK JOURNALS REQUIRED AND FOR SALE

If any subscriber can spare copies of the January 1945 and June 1950 issues of the *Journal of Laryngology & Otology* they would be very much appreciated by the Librarian of The National Institute for the Deaf, 105 Gower Street, London, W.C.1.

If anyone would like to buy a set of the Journals from 1921 to 1960 (mostly bound volumes), and the two relevant indexes, etc., please apply to Donald G. P. Macalister, 5 Clarendon Place, Leamington Spa.

THE MOUNT SINAI HOSPITAL

An intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given January 13th, 1962 to January 26th, 1962 by Dr. Irving B. Goldman and staff at the Mount Sinai Hospital, New York in affiliation with Columbia University.

Candidates for the course should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, Fifth Avenue and 100th Street, New York 29, N.Y.

General Notes

CORRECTION

In the June 1961 issue of *The Journal of Laryngology & Otology* in the article by Professor F. C. Ormerod on "The Metabolism of the Cochlear and Vestibular End-Organs", the first paragraph on page 566 should read:—

Poulson (1959) and Jensen and Vilstrup (1960) have shown also that the proportion of mucopolysaccharide is greater in the perilymph of the superior parts of the labyrinth — the utricle and semicircular canals — than in the inferior parts — the cochlea and saccule — of the cod fish. They are of the opinion also that all the mucopolysaccharide in the superior part of the cod's labyrinth is hyaluronic acid.

EXAMINING BOARD IN ENGLAND BY THE ROYAL COLLEGE OF PHYSICIANS OF LONDON AND THE ROYAL COLLEGE OF SURGEONS OF ENGLAND

Diploma in Laryngology and Otology

The Secretary reports to the two Royal Colleges that at the First Part of the Examination held in June, 1961, 61 Candidates presented themselves, of whom 32 passed; and that at the Second Part of the Examination held in June, 1961, 33 Candidates presented themselves, of whom 22 passed.

The following 19 Candidates, having complied with the regulations of the Board, are eligible to receive the Diploma, viz.:

Ahmadizadeh, Seyed Majid	2 Azimi Street, Teheran, Iran.
Al-Nawab, Dhia	291 Golders Green Road, N.W.11.
*Anklesaria, Darius Manek	c/o Wilson, 22 Gilmore Place, Edinburgh 3.
*Araj, Jabra Saba	Beit Jala, H.K. Jordan.
Evans, John Noel Gleave	2 Kingswood Road, Shortlands, near Bromley, Kent.
Gathercole, Raymond	c/o Military Hospital, Colchester, Essex.
Ghanekar, Dinkar Govind	Nuffield Hostel, Royal College of Surgeons, Lincoln's Inn Fields, W.C.2.
Jafferey, Asaf Husain	Hackney Hospital, E.9.
Kerawalla, Sam Dinshawji	17 Crawford Road, S.E.5.
Mahindrakar, Naganath Hanamanthrao	Royal Infirmary, Bolton, Lancs.
Maung, Thein	Croydon General Hospital, Croydon, Surrey.
Murugendrappa, Togaleri Revappa	c/o M. S. Nanjappa, 1890 Wesley Road, Tilak Nagar, Mysore, India.
Noel, Joseph Gerard Maxime	159 Cromwellsfort Road, Crumlin Cross, Dublin 12.
Penn, Alan Laurence	167 Adelaide Road, N.W.3.
Roy, Nripendra Nath	Royal Berkshire Hospital, Reading, Berks.
Shankara, Mahanta Devaru	2884 Behind the Law Courts, Mysore, India.
Sil, Kamalesh Chandra	Royal National Throat, Nose and Ear Hospital, Golden Square, W.1.
Singh, Sukh Dev	c/o Royal National Throat, Nose and Ear Hospital, Gray's Inn Road, W.C.1.
Tembe, Dinkar Parashuram	24 Augustus Road, Edgbaston, Birmingham, 15.

* Examination passed previously.

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